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ABSTRACTS

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Influence of surgery on renal amyloidosis. *H. Kaaroud, F. Ben Moussa, R. Goucha, E. Abderrahim, F. Ben Hamida, T. Ben Abdallah, F. El Younsi, A. Kheder, H. Ben Miaz, Department and Laboratory of Nephrology, Charles Nicolle Hospital, Tunis, Tunisia.* It is well known that renal amyloidosis (RA) leads to ESRD in a few years. This evolution may be accelerated by several factors such as steroids, renal vein thrombosis, infections or surgery. We report 22 patients (14M,8F) mean age = 41.6 years (13-72) with RA in whom surgery revealed or aggravated renal disease. The group I includes 15 patients with no previous history of renal disease and who developed oedema few days after surgery with acute renal failure in 5 of them. Proteinuria was present in all the cases with a nephrotic syndrome in 10. Percutaneous kidney biopsy (KB) showed renal amyloidosis in all patients (AA+ = 8 cases, AA− = 3 cases). Only 9 patients were followed-up (mean period = 40 months): 2 patients are stationary; 1 is on complete remission 2 are on HD and 4 died. The group II includes 7 patients with a previous history of nephropathy (Histologically proven amyloidosis: 3 CRF = 1, Oedema: 3). All these patients developed oedema few days after surgery with acute RF in 4 patients. KB performed in all of them showed RA (AA+ = 33, AA− = 1). 6 patients were followed up for a mean period of 11 months: 5 died, 1 patient is on HD. The influence of surgery on renal amyloidosis is often unforeseeable. It may have no effect on renal disease, but very often it reveals RA and sometimes dramatically aggravates the course of the disease with occurrence of irreversible CRF. The pathogenic role of surgery on RA is discussed.

Cytoskeletal proteins differentiate between progressors and non progressors in treated idiopathic membranous nephropathy (IMN). *N.A. Tamimi[1,2], P.E. Stevens[1], P.L. O'Donnel[3], P.G. Strange[2], E.C. Muchaneta-Kubara[4], A.M. El Nahas[4], Canterbury Hospitals NHS Trust[1], UK.* Cytoskeletal proteins such as α-smooth muscle actin (α-SMA) and vimentin (V) have been associated with renal fibrosis, injury and scarring. This study was aimed at defining the role of these markers in IMN. We studied 21 patients with IMN treated by immunosuppression attempting to define the role of α-SMA and V positive cells in this form of nephropathy. There were 15 non-progressors (NP) and 6 progressors (P). The clinical, histological and immunohistochemical (IHC) characteristics of both groups were compared. Diastolic pressure was significantly higher during follow-up period but at presentation in patients with progressive disease ($p = 0.007$). The degree of proteinurina, severity of interstitial infiltrate and fibrosis did not differentiate P from NP. However, vascular sclerosis was more severe in P compared to NP ($p = 0.0053$). Glomerular α-SMA was significantly higher in P ($0.363 \pm 0.022\%$) than in NP ($0.172 \pm 0.07\%$) $p = 0.003$. Interstitial α-SMA was also higher in P but did not reach statistical significance while interstitial V was higher in P ($0.207 \pm 0.07\%$) compared to NP ($0.087 \pm 0.049\%$) $P = 0.0019$. Vimentin was detected in the tubules as well as interstitial cells. The expression of glomerular α-SMA and interstitial V may be useful prognostic implications as they appear to differentiate between patients with IMN who respond to immunosuppression and those who continue to progress.

Post-dysenteric haemolytic uraemic syndrome in children during an epidemic of shigella dysentery in Kwazulu/Natal. *R. Bhimma, N.C. Rollins, H.M. Coodavia, M. Adhikari, Department of Paediatrics and Child Health, University of Natal, Durban, South Africa.* Post-dysenteric haemolytic uraemic syndrome (HUS) following E. Coli 0157:H7 infection is the most widely prevalent and best described form of the disease. However the syndrome following Shigella dysenteriae type 1 (SD) infection has been only sketchily documented. A new epidemic of SD1 has changed the epidemiology of HUS in Southern Africa. In this region HUS was endemic but restricted to mainly White children in the northern provinces of South Africa and in Zimbabwe. We report on 81 of 107 cases of HUS, during the period July 1994 to February 1996, following an outbreak of SD1 dysentery in Kwa Zulu/Natal. All patients, excluding 1 child, were Black with a mean age of 38 months (range 1–121) and 50 (61.7%) were males. The mean duration of dysentery was 11.3 days (range 1–41) and that for HUS was 15 days (1–91). The majority of patients had acute oliguric renal failure (90.1%); 42 (51.6%) required peritoneal dialysis. Complications included encephalopathy 30 (37.0%); convulsions 12 (14.8%) and hemiplegia 2 (2.3%); gastrointestinal-perforation 8 (9%); protein losing enteropathy 26 (32.1%); toxic megacolon 4 (4.9%); rectal prolapse 5 (6.2%); congestive cardiac failure 3 (3.7%); cardiomyopathy 3 (3.7%); infective endocarditis 1 (1.2%); septicaemia 15 (18.5%); and disseminated intravascular coagulation 7 (8.6%). Leukaemoid reactions in 74 (92.2%) patients; hyponatraemia in 56 (70%) and hypoalbuminaemia in 67 (87.1%) were characteristic laboratory findings. Stool culture for SD1 was positive in only 7 (8.6%) patients; shiga toxin assays were not undertaken. Outcome was as follows: recovery 32 (39.5%); impaired renal function 8 (9.9%), chronic renal failure 26 (32.1%); end stage renal failure 1 (1.2%) and death 14 (17.3.%). The high mortality and morbidity in SD1-associated HUS presages the need for the development of novel therapy in the management of these patients, which can be used in developing countries.

HCV in renal failure. *Maher Ramzy, Cairo University, Egypt.* **Hepatitis C Associated Glomerulopathy:** Glomerulopathy often accompanies chronic liver disease although frequently it is clinically silent (Newell GC 87). Both cryoglobulinemic and non cryoglobulinemic GN were described. Direct evidence for renal deposition of HCV related antigen has never been presented except recently (Okada K 96). We studied twenty patients, with HCV chronic liver disease (biopsy proven) and proteinurial 2gm 24th (data will be presented). Interferon alpha had been tried (Johnson R 94). The optimal treatment strategy, however, remains to be defined. *HCV in chronic patients*: **Risk factors in dialysis patients:** Blood transfusion, nosocomial transmission was minimized by screening of blood donors and Erythropoietin 1.1. Isolation of HCV positive patients in a separate unit is controversial. Following universal hygiene precautions (CDA-Atlanta) keeps seroconversion very low. Vaccination and Immune serum globulin: Vaccine is unexpected in the near future due to multiple serotypes. Immune serum globulin has not yet proved effective (Marian 1.92). Treatment of chronic HCV in

dialysis patients: Interferon therapy is the only effective treatment. A sustained response of greater than 30% can be achieved (Davis JL 94). *HCV in renal transplantation*: **Incidence:** Is mainly determined by the rate of infection before transplantation (TP), HCV infection can be transmitted by renal grafts. The rate differs between studies (Roth D 92 Vincenti F 93). **Expression and evolution of HCV infection after renal transplantation:** Anti HCV antibodies do not disappear after renal transplantation. The biological signs of the disease are in short term basis (Stempel CA 93: Roth D 94). Immunosuppressive treatment of renal transplant patient is likely to activate viral replication. It is possible that Ch liver disease affects the life or renal transplant recipients at a later stage. The uncertainty does not appear as a contraindication of transplantation in non-cirrhotic HCV-ve patients (Goffin R 95) There is no effective therapy for Tx recipients with HCV till now.

Dialysis outcomes. *Nathan Levin, Renal Research Institute, New York.* It is conventional wisdom that gross and adjusted annual mortality of Americans on dialysis greatly exceeds the analgous rates reported in many other countries. Many explanations have been offered for the U.S. findings including higher acceptance rates of older patients with greater comorbidity; genetic factors; dialyzer reuse with noxious or ineffective chemicals or resulting in dialyzers with much reduced function; under nutrition of patients; failure to deliver an adequate dose of dialysis in the majority of patients, and less direct physician's care; an alternative explanation is that in some countries the reporting of mortality and of comorbidity is incomplete. The role of the dialysis procedure itself, including membranes, lines, endotoxins in water and dialysate, vascular access problems, subclinical infection and chronic inflammation all require elucidation. Evidence of the presence and importance of increased oxidative stress is controversial. The influence of background cardiovascular illness in the general medical community as affecting dialysis mortality and hospitalization is currently receiving attention. A major question is whether earlier initiation of dialysis with better management of early cardiovascular damage will make an impact on the life expectancy of dialysis patients. Increasing attention is being paid now to measure of quality of life and the effect of psychosocial factors. Studies of reasons for patients' choice of modality, dropout from peritoneal dialysis (particularly), non compliance and decisions to stop treatment are now underway. The use of quality and outcome indicators for credentialling and to competitively assess dialysis facilities for managed care has become a major trend in the U.S.

Iron physiology and metabolism in CRF. *I. Cavell. University of Wales College of Medicine, Cardiff, UK.* Iron metabolism is dominated by the demand for iron from developing erythroid cells. This is a highly dynamic process resulting in the release of 2–3 million red cells per second and requires 30 mg iron per day. Of the 4 g of iron in a normal human, some 3 kg is located within the circulating red cell mass. This is the single biggest store of iron in the body. At the end of each red-cell's lifespan iron within it will be released and become available for new red cell synthesis again. A further 1 g of iron will be deposited in the reticuloendothelial iron stores. This will largely be in the form of haemosiderin. The rate at which the iron within this insoluble material can be made available for red cell synthesis can be a limiting factor. The plasma transferrin pool accounts for some 4 g of iron in a normal subject. The iron in this pool is extremely labile and turns over 10-12 times a day. The concentration of in this pool is determined by the transient balance struck between in-flow and out-flow. The first is the measurement of iron in the erythroid pool, the second is the measurement of iron in the store, the third is an assessment of the adequacy of iron delivery to developing erythoid tissue. Functional iron deficiency is a new concept that has emerged from the use of erythropoietin in patients with chronic renal failure. It is represents a failure of the rate of iron supply to support erythropoiesis. This may occur even when there are adequate amounts of iron in the stores. When iron supply is adequate each new red cell will contain some 30 pg of haemoglobin. When iron supply is limiting, cells will emerge with suboptimal amounts of haemoglobin within them. When the individual cell haemoglobin concentration is 28 g/dl it is deemed to be hypochromic. The percentage of hypochromic cells in the circulation is a direct measure of the adequacy of iron supply to the erythroid tissue. In patients with the anaemia of chronic disorders, including those with malignancy, it is usual to find raised iron stores and raised serum ferritin concentrations. This has been mistakenly attributed to some inability of the reticuloendothelial cells to release iron (the R E block). In reality the anaemia of chronic disorders is the result of suppression of erythroid activity as a direct result of the inflammatory cytokines associated with the disease. In consequence the iron that had been circulating in the red cell pool cannot be reincorporated into new red cells and is deposited in the iron stores. The serum ferritin concentration reflects the increasing level of these stores: it is raised as a consequence of the anaemia. Erythropoietin therapy can overcome this erythroid suppression. When it does, the need for additional iron supply to the erythroid tissue will be the same as that seen in renal patients. Functional iron deficiency will supervene when that supply is inadequate. The need for the rapid supply of iron in the correction phase of EPO therapy will be the same in these patients as it was in those with renal failure. Iron supply will remain the key to the successful and efficacious use of recombinant human erythropoietin in these patients

From membrane technology to therapy outcome. *G. Brown, J. Vienken, Fresenius Medical Care, Germany.* Membrane manufacturing technology influences the physical and chemical properties of a membrane, its biostability and the degree of extractables. The chemical properties depend on the base polymer. But even selection of the same base polymer can lead to different therapy outcomes due to introduction of surface modifying groups or as a consequence of copolymerisation reactions. Established examples are the modified cellulose membranes Hemophan and the cellulose acetates. Today extensive membrane development is focussed on modification of synthetic polymers such as polysulfone (Psu) which has become established as a highly blood compatible and efficient membrane for the treatment of ESRD. Investigations of several Psu membranes have uncovered significant differences in physiocochemical properties and therapy outcome which may be related to membrane productin technology and sterilisation influences. Advanced membrane formation technology additionally offers the manufacturer the possibility to specifically adjust membrane permeability to suit the prescribed therapy. Low-flux membranes can be more readily manufactured from the polymers already established for renal therapy but limit blood purification potential. Highly permeable membranes enable elimination of larger toxins especially when high fluid fluxes for efficient convective transport across the membrane are applied. Such membranes impose high requirements on the membrane manufacturer and on the dialysis centre with regards to such as the microbiological quality of water and dialysis fluid.

Peritoneal dialysis overview. *Dr. Ram Gokal, MD, FRCP, Royal Infirmary, Manchester, UK.* Peritoneal dialysis is now an established form of dialytic therapy accounting for about 15% of the global dialysis population. Its use varies depending on local finances, resources and physician biases. Patient survival is equivalent to that on haemodialysis but technique survival is inferior, whilst long term PD (greater than eight years) is limited to a small fraction of patients starting this therapy. The main reasons for this drop out are peritonitis, peritoneal access, inadequate dialysis and patient psychosocial factors. These areas have received considerable attention in the last decade. There have been great strides in the understanding of peritoneal physiology, host defence, solute and fluid transport and a means to minimise damage to the peritoneum (eg newer physiological solutions). There has been a decline in peritonitis and access related problems with the use of disconnect systems and better catheters and insertion techniques. Currently there is enormous interest in the adequacy of dialysis and how the targets for these impact on outcomes (survival, hospitalisation and nutrition). Patient's quality of life appears to be better than on haemodialysis whilst the cost of CAPD in the West is lower than that of HD). Whilst PD has limitations its use in developing countries is likely in increase. PD has to be integrated with other renal replacement therapies for best patient outcome.

Peritonitis—Pathogenesis, prevention and treatment. *Dr. Ram Gokal, MD, FRCP, Manchester Royal Infirmary, Manchester UK.* Peritonitis

still remains a major cause of treatment failure and hospitalisation in patients on peritoneal dialysis. It results in a marked increased in effluent protein loss, and repeated attacks may lead to a hypermeable membrane, loss of ultrafiltration and poorer outcome. **Pathogenesis:** Contamination at the time of PD exchanges still remains a major cause, but about 15-20% of episodes are secondary to catheter infection. Most of these episodes (70%) are secondary to gram positive organisms. In the absence of known contamination, peritonitis due to gram negative organisms is considered to be enteric in origin. Host defense mechanisms are important but no direct link has been established with opsonins or cellular functions. **Prevention:** Touch contamination related episodes (staph epidermidis) have decreased considerably by improvements in connection technology. Prevention by boosting host defence and vaccination has not been successful. Careful training, attention to details, reinforcement and good catheter implantation are important strategies for prevention. **Treatment:** Initially emperic therapy of peritonitis is with intra peritoneal antibiotics. A recent expert committee report recommends against Vancomycin as a first line antibiotic (because of the emergence of Vancomycin resistant enterococci) and advocated first generation Cephlosporins with an aminoglycoside. The problem area still remain infections with staph aureus, pseudomonas and fungi where the outcome is poor and catheter removal may be necessary to impact a cure.

Physicochemical aspects of kidney stone disease. *A.L. Rodgers, Chemistry Department, University of Cape Town.* Since urolithiasis involves the precipitation of salts from a saturated solution, physiocochemical principles are applicable. Research focuses on two main areas: stone composition, structure and ultrastructure and crystallization mechanisms. Two "fingerprint" methods—infrared spectroscopy and x-ray powder diffraction—are used for unequivocal identification of stone composition which is useful for diagnosis and treatment. Application of the concept of epitaxy permits the design of molecules which can block growth sites. Scanning electron microscopy has shown that stone matric acts as a binding agent in the formation of crystal aggregates. The metastable limit and crystallization kinetics of a given urine provide a measure of its stone forming potential. Sophisticated experimental system (eg the continuous crystallizer) distinguish between nucleation, growth and aggregation mechanisms. Techniques for counting and sizing crystals (eg Coulter Counter, Malvern Particle Sizer) have shown that aggregation is probably the crucial step that needs to be targeted by potential inhibitors. Urinalysis data have been used to develop powerful mathematical expressions which provide quantitative estimates of the risk of stone formation. These include physiocochemical risk ratios, quotients and indices (eg relative supersaturation, activity products) which are useful for assessing the efficacy of therapeutic regimens.

Ethics & economics of treatment of renal failure in Africa (Sudan). *Suleiman, S.M.* Sudan is a million sq. miles in central Africa over the Equator, having borders with 9 African countries and a 10th border on the Red Sea. Population about 36 million. Transport, communications, socio-economic conditions are rather poor over most parts with high prevelance of most endemic diseases known to affect the kidneys. Facilities for dialysis: 3 PD & 4 HD (governmental centres) and 2 private centres in Khartoum Port Sudan (over the Red Sea). Incidence of ARF pmp (unknown), CRF pmp (estimated 70–140). Aetiology of ARF: Dehydration (GE) in children, in adults: P.falciparum malaria, hair-dye poisoning, septicaemia (septic abortions) and snake bites. CRF: commonest causes G/N (idiopathic and secondary) Renal stones ± infections uncontrolled DM, BP. Emergency PD is done on available beds. Selection for chronic dialysis, young with a suitable donor with a possibility for Tx, ARF has got priorities. Funding is mainly governmental with help from national societies (2) alms from some persons donations & help from individuals and societies in other countries.

Renal failure in Africa. *Youmbissi T. Joseph: Cameroon.* The region of Central Africa has a population of about 50 million people and presents a great diversity in its geography and economy. There is no reliable data concerning the incidence and prevalence of Acute Renal Failure but in Cameroon, medical causes predominate (Enteric disease + non enteric sepsis). The incidence of chronic renal failure is not less than 150 per million per year and Immune complex GN are by far the commonest causes with severe uncontrolled hypertension, the second. All treatment modalities for ESCRF are available albeit on a very small scale. Besides the costs, maintenance, laboratory and technology problems mar the implementation programmes. There is no structural funding for the care of renal patients and it is done on a fee for service basis. The subsequent ethical problems are immense. Preventative neprholgy seems to be the only way out as well as horizontal and vertical co-operation.

Diabetes and coronary heart disease (CHD): Hypertension, genetics and microalbuminuria. *Estacio R.O., Schrier R.W., University of Colorado Health Sciences Center, Denver, Colorado, USA.* Diabetes is responsible for a significant number of deaths per year with nearly half of the deaths are secondary to heart disease. Studies have shown that diabetics are 2–4 times as likely to die from heart disease as people who do not have diabetes. The present study in non-insulin dependent diabetes evaluates various cross sectional baseline patient characteristics from the ongoing Appropriate Blood Pressure Control in Diabetes (ABCD) Trial and its association with CHD. We evaluated the possible associations of glycosylated hemoglobin, insulin use, hypertension (systolic blood pressure >140 mmHg or diastolic blood >90 mmHg), urinary albumin excretion and the effect of the angiotensin converting enzyme (ACE) genotype with CHD. CHD was found not to be associated with insulin therapy, glycosylated hemoglobin or the ACE genotype but was associated with hypertension (OR 1.39, 95% CI 1.00–1.93), and overt albuminuria (OR 1.59, 95% CI 1.03–2.46). We also found that the presence of the DD genotype of the ACE gene was associated with an increase in left ventricular mass index (parameter estimate 9.90 gm/height $(m)^2$, 95% CI 3.74–16.07). In summary, the presence of hypertension and diabetic nephropathy as represented by overt albuminuria are associated with the presence of CHD. Study also demonstrates an independent association between the presence of the DD genotype and an increase left ventricular mass which has been demonstrated to be a predictor of cardiac mortality.

Peritonitis in continuous ambulatory peritoneal dialysis (CAPD): Prevalence in a single center. *Ben Hamida F., Ben Abdallah T., Abdelmoula M., El Younsi F., Abderrahim E., Goucha R., Ben Moussa F., Ben Maiz H., Department of Nephrology and Internal Medicine (Pr H. Ben Maiz) Charles Nicolle Hospital. Tunis-Tunisia.* Despite technical improvements of peritoneal dialysis over the last ten years, peritonitis remains the major complication and the most common cause of technique failure and patient morbidity. We report our experience in patients with CAPD peritonitis. It is a retrospective study including 145 peritonitis occuring in 69 patients (31 males and 38 females) between January 1991 and February 1997. 74% of patients were autonomous and 26% were helped by a member of their family when they manipulated the catheter. During the period of study, 88.4% of patients presented 1 to 3 peritonitis and 11.6% presented 4 to 7 peritonitis, with an average of 2.1 peritonitis per patient. The mean delay between peritonitis and the begining of treatment by CAPD was 15.5 months (1 to 188 months). Organisms isolated in peritoneal fluid were: 50 (34.5%) cocci gram positive, 28 (19.3%) bacilli gram negative and 3 (2.1%) bacilli gram positive. Organism culture was negative in 61 (42.1%) of cases. There are no difference between this bacteriological spectrum and the one observed in a previous study realized from 1986 to 1990. All patients received medical treatment: intraperitoneal, intravenous or oral antibiotics. The outcome was favourable in most cases. In conclusion, CAPD peritonitis were frequent, occured mainly after an aseptic manipulation of the catheter. Antibiotics initiated prior to isolating the organism must be active against staphylococcus and bacilli gram negative.

An evaluation of the Banff Classification. *W.D. Bates, D.R. Davies, K. Welsh, D.R. Gray, S. Fuggle, P.J. Morris, Oxford Transplant Centre—Tygerberg Hospital—Stellenbosch University.* This study was done on 383 consecutive transplants performed between 1985 and 1989, 351 with graft followup to 5 years. Regarding diagnostic value, use was

made of a previous study of a subgroup of this cohort in 1987–1988. A pre Banff sensitivity for the diagnosis of acute rejection of 75% (24/32) was derived with a specificity of 87% (88/101). With Banff classification the sensitivity and specificity were both marginally lower—73% (16/22) and 85% (79/93). Of note was the large borderline group—48/181 (27%) of the suitable biopsies. Concerning prognostic value, the most severe biopsy in the first 35 days was correlated with graft outcome at 3 months, 1, 3 and 5 years. Biopsies were available on 293 of the 351 patients (83%). Groups as follows: Normal or non-rejection—60/293 (20%); Borderline—(34%); AR I—(18%); AR IIA—(6%); AR IIB—(14%); AR III—(1%); AR IIIC—Necrotising vasculitis—(3%); Widespread necrosis (? AR III)—(3%). Regarding outcome the last 2 groups had a very poor prognosis—18% at 3 months, 6% at 5 years. The other vascular rejection groups, IIB and III, had an intermediate outcome—78% at 3 months, 61% at 5 years. An interesting finding was that the other 4 groups (normal, borderline, and cellular AR I and AR IIA) had not only the predictably best outcome—95% at 3 months but that at 5 years all 4 had almost identical graft survival of 78%. **Conclusions**: Banff has diagnostic value with moderate sensitivity and specificity but the large borderline group is one unresolved area. Prognostically 3 groups emerge—severe necrotising vasculitis or widespread necrosis leads to very frequent early loss, other vascular involvement leads to moderate early loss, with slower loss out to 5 years while the normal, borderline and usually reversible forms of cellular rejection show steady decline over 5 years with similar 5 year outcome. These 3 groups are very similar to the NIH-CCTT 3 types of acute rejection.

Patient function and well-being on CAPD. *A.Z. Muranda, S. Naicher, L. Botden, G. Nel, Addington Hospital and University of Natal, Durban, South Africa.* Dialysis outcomes underemphasise function and well-being, while the nutritional and mortality benefits are well established. **Aim**: to assess the effect of adequate dialysis on patient well-being. **Method**: adequate dialysis was defined as an 'Adequest' urea clearance, KT/V > 2.1. Well-being was assessed by a structured questionnaire with graded responses. **Results**: 82 patients were eligible, all functionally anephric.

	KT/V < 2.1 (n = 55)	KT/V 2.1 (n = 27)
Age *years*	39.7	40.6
CAPD *months*	46	31
Hb/g/dL	8.5	8.8

In the underdialysed group, the odds ratio for loss of gainful employment was 1.5, nausea 7.3, vomiting 9, asthenia 4.4, unsatisfactory sex 1.4, insomnia 1.2, restless legs, 0.2 and poor coping 1.8. **Conclusion**: Adequate dialysis improves patient function and well-being. This focusses rehabilitation at outcome types that are important to patients and families' activities of daily living.

Acute peritoneal dialysis in children. *R.D. Gilbert, Red Cross War Memorial Children's Hospital, Private Bag Rondebosch 7701, W Cape.* Fifty-two children underwent acute peritoneal dialysis (PD) between April 1995 and March 1996. Their ages ranged from 2 days to 14 years. The causes of renal failure were: acute tubular necrosis (ATN) 35, haemolytic-uraemic syndrome 7, tumour lysis syndrome 5 and miscellaneous 5. The causes of ATN were diarrhoea 7, sepsis 19, post cardiac surgery 3 and cardiac abnormalities without surgery 4 and toxins 2. Twenty-five patients were initially dialysed using acute "stick" type catheters, 15 using catheters inserted using a guide wire and 12 with Tenckhoff catheters, 6 were of which were placed using a peel-away sheath technique. Twelve catheters required revision. The overall mortality was 52%. Mortality was higher in those dialysed in intensive care units (75%) than in the renal ward (0%, $p < 0.02$), reflecting multiple organ system involvement in these patients. Six of seven children under a month died. This was not significantly different from those over 1 month. PD can be performed on almost any child. The mortality rate in children with renal failure and multiple organ system failure is extremely high. ATN secondary to sepsis and diarhoea is the most frequent cause of ATN requiring dialysis in our hospital.

Peritoneal dialysis in the treatment of acute renal failure: A Congolese experience. *Assounga A.G., Mpio I., Assambo-Kieli C., Ngakosso N., Brazzaville's University Hospital and Marien Ngouabi University, Brazzaville, Congo.* Management of acute renal failure is still posing major problems in Africa. Peritoneal dialysis, a renal replacement therapy relatively easy to introduce, can be very useful. This is a report of a retrospective study of the use of peritoneal dialysis in the treatment of acute renal failure in Brazzaville's University Hospital from January 1990 through September 1996. Records of patients hospitalized for ARF (192 cases) were examined and all patients treated with peritoneal dialysis (18 cases = 0.9%) were included. Peritoneal dialysis was the only replacement therapy available. All patients with ARF needing dialysis were put on peritoneal dialysis, except those having a recent abdominal surgery. The continuous ambulatory peritoneal dialysis (CAPD) method was applied using a rigid catheter in 12 cases and a Tenckhoff catheter in 6 cases. A lactate containing dialysis solution was used. A complete recovery of kidney function was observed in 11 cases, chronic renal failure in 4 patients and death in 3 cases. Thus peritoneal dialysis, easy to use, may help save lives in many centers, and especially in those where hemodialysis is not available.

Renal transplantation in the management of end stage diabetic nephropathy. *V.K. Rao, University of Minnesota Medical School, Minneapolis.* Diabetic patients now comprise 30% to 40% of dialysis and transplant population in the United States. There is no ideal test that will predict the future risk of developing diabetes in the renal donor. We use the rapidity and magnitude of Beta Cell insulin release following an intravenous glucose load to screen the family donors. All potential candidates undergo dobutamine stress echo cardiography and Doppler assessment of peripheral vasculature and carotid arteries. Occlusive vascular disease continues to progress in these patients leading to much of the morbidity and mortality in the post transplant period. Compared to non diabetics, diabetic transplant patients have higher incidence of coronary and peripheral vascular disease. Approximately 17% undergo amputations for ischemic disease. Retinopathy and neuropathy remain stable in the majority of transplanted patients. Infection risk appears to be high, compared to the non diabetics but the rejection rate is similar. Cardiovascular events account for the majority of deaths, particularly in the second decade after renal transplantation. Of the different risk variables, recipient age, donor source, cyclosporine immunosuppression, type II diabetes and pre-existing clinical vascular disease have an independent effect on survival results. The risk of graft loss from chronic and acute rejection is similar to the non diabetic patients. Diabetic vascular lesions are frequently observed in the transplanted kidneys but the incidence of graft loss from recurrent disease is less than 5%. It is gratifying to note that 60% of diabetic renal transplant patients can attain full rehabilitation; 30% partial rehabilitation and only 10% are medically disabled.

Hypertension in Africa: Its impact on the kidney. *O.O. Akinkugbe, University of Ibadan, Nigeria.* Hypertension is today, indisputably, the most common cardiovascular condition, and indeed the most endemic non-communicable disorder on the African continent. Its prevalence in both rural and urban communities varies from 5 to 15 per cent of the adult population, and the risk factors associated with its genesis are much the same as in Western societies. The kidney is both a prime cause and a vulnerable target. Renal complications occur in a significant percentage of young adults with severe hypertension. However a substantial proportion of African hypertensives will already have had one form or other of glomerulonephritis, and in such cases hypertension accelerates the unrelenting progression to renal failure. The nephrotic syndrome is a common pathway and its aetiologic profile in Africa differs in some respects from that in Western Communities. Many hypertensive renal failures seen for the first time often present with end-stage shrunken kidneys, at which point it becomes impossible to unravel the pathogenesis—hypertension or glomerulonephritis, a "hen or egg" dilemma. The role of bacterial and protozoal nephropathy (Malaria, Schistosomiasis) will be briefly discussed. Programmes are now being put in place in a number of African countries to address the emerging non-communicable disease (NCD) before their devastating effects take centre stage. The recent Nigerian experience on NCD

survey and strategies for control will be presented. The prohibitive cost of renal replacement care in the African context dictates that preventive strategies be given greater prominence in our efforts to confront the challenge of hypertensive renal disease in Africa.

Hypertension: Recent advances. *Omar Abboud, University of Khartoum, Sudan.* Angiotensin (AT) II is produced in plasma by the sequential processing of circulating angiotensinogen by renin and AT I converting enzyme (ACE). The enzyme renin, produced by the kidney, is the major regulating factor of AT II production in blood. Local AT II production has also been described in several tissues where the conversion from AT I to AT II is done by local enzyme independent of the control of ACE. The role of this tissue angiotensin system is being more appreciated recently. Its possible inhibitors will be discussed including the new group of AT II receptor blockers. Further developments in the renin angiotensin system (RAS) are the recognition of angiotensin 1-7 which has a vasodilator effect antagonistic to the action of AT II. Also with the recent ability of cDNA cloning of ACE, it was possible to study the ACE gene. A frequent insertion/deletion polymorphism characterized by the presence of a 287 bp fragment situated in the intron 16 of the ACE gene. It could have a predictive value on the therapeutic effect of ACE inhibitors on proteinuria. On the treatment aspect microalbuminuria is now considered as important in hypertension as it is in diabetes in detecting early renal damage. It can play a role in the initiation of hypotensive therapy in borderline cases with no other evidence of target organ damage. The target level of blood pressure control to be achieved by hypotensive therapy will be dicussed with its implications of the reduction of the incidence of CVAs and ischaemic cardiac events. Nitric oxide is emerging as an attractive therapeutic agent for hypertension being recognized as the mediator for many vasodilator substances. A focus on its actions and therapeutic potential will be made.

IgA Nephropathies. *Rashad S. Barsoum, Cairo University. Egypt.* IgA nephropathy (IgAN) is the commonest form of glomerulonephritis (GN) worldwide, albeit being quite rare in Africa and other areas. Primary IgAN may be encountered as an isolated mesangioproliferative GN (Berger's disease), associated with cutaneous vasculitis and arthritis (Henoch Schonlein syndrome—HSS), or it may overlap with other renal disorders as minimal change disease, membranous nephropathy, Wegener's granulomatosis or Thin Basement Membrane disease. Males are more susceptible, with a peak age of onset around the twenties, except with HSS, which starts one decade earlier. Hematuria is the predominant symptom, being gross, painless and intermittent in one third of cases. In another third, it is microscopic and often associated with persistent proteinuria. The remaining third of patients present with hypertension, chronic renal failure, acute nephritic syndrome, or acute renal failure. The main laboratory features include the characteristic urinary findings, elevated serum IgA in 35–50% of cases and the presence of circulating IgA immune complexes, rheumatoid factor and/or fibronectin aggregates. Urinary IL-6 is increased, correlating with disease activity. Renal biopsy shows focal or diffuse mesangial cell proliferation, widening of the mesangial matrix and variable crescent formation, which is the main criterion for histological classification into 6 grades. Immunofluorescence characteristically shows polymeric IgA_1 mesangial & capillary loop deposits, together with IgG, IgM, C_3 and other complement components of the properdin pathway. EM shows the mesangial & subendothelial deposits. the course of primary IgAN is fairly benign, with a renal survival of 80% at 10 years and 70% at 20 years after the onset. There is no specific treatment, but response to steroids is usual in minimal change disease with IgA deposits, common with HSS and reported with crescentic IgAN. Some benefit has been obtained with ACE-inhibitors, fish oil and intravenous IgG. Secondary IgAN is reported with chronic liver disease, celiac disease, dermatitis herpetidiformis, several seronegative rheumatoid diseases, familial Mediterranean fever, hematological malignancies, AIDS and many other conditions. In most of these conditions, the disease is mild or asymptomatic. A notable exception is hepatosplenic schistosomiasis, where IgA glomerular deposits hallmark a severe and progressive disease. The pathogenesis of IgAN is complex. Suggested mechanisms include abnormal IgA or its circulating forms, switching of peripheral blood mononuclear cells to predominant IgA response and specific glomerular susceptibility. It appears that schistosomal glomerulopathy satisfies the pre-requisites for the induction of progressive glomerular pathology, by providing a "switching" mechanism as well as "priming" the glomeruli for subsequent IgA deposits by prior deposition of immune complexes involving parasitic antigens.

Management of renal failure in central Africa in 1997. *Assounga A.G., Brazzaville's University Hospital and Marien Ngouabi University, Brazzaville, Congo.* Sub-saharian Africa is lagging in the development of nephrology in Africa. In this report, we examine the current status of the management of kidney failure in the region. This is based on a review of publications on nephrology in the region. The central Africa region includes: Angola, Burundi, CAR, Congo, Democratic Republic of Congo, Gabon, Rwanda and Tchad. With the population of almost one hundred people, and between 30 and 40 patients reaching the end stage of kidney failure per million, per year, the region has less than 20 trained nephrologists practicing in 6 nephrology centers. Renal conservative treatment is offered in all centers. Renal replacement therapy is available in 3 countries: Hemodialysis is used in 2 centers while peritoneal dialysis is practiced only in 2 centers. No kidney transplantation is currently practiced in the region, but patients transplanted abroad are currently following their immunosuppressive treatment in the region. Obviously, a lot needs to be done both in nephrology training and equipment, in order to advance the level of basic care of kidney failure in Central Africa. Strengthening the few existing nephrology centers and creating new ones may be the way to go.

Renal failure in Africa. *Anthony J.O. Were, Nairobi, Kenya.* This report looks at Renal diseases in Sub-Saharan Africa with particular emphasis on the pattern of renal diseases in East Africa. The causes of acute renal failure range widely from acute glomerulonephritis due to acute post Streptococcal Glomerulonephritis to the fluid losses in cholera. Haemorrhage in Obstetrics, in Schistosomal and Portal Hypertension, and in massive road traffic accidents are highlighted as important causes of acute renal failure. Malaria associated renal failure is a frequently recognised cause to which clinician in the region must be alert. An approach to management in a District hospital setting is suggested. The grave implication of development of chronic renal failure and end stage renal disease, principally from chronic glomerulonephritis is presented against the scarce availability of renal replacement therapy in the region.

Renal failure in the southern zone of Afran. *C.R. Swanepoel, Department of Medicine, Renal Unit, Groote Schuur Hospital.* The southern zone of AFRAN includes Mozambique, Malawi, Zambia, Angola, Zimbabwe, Botswana, Namibia, Lesotho, Swaziland, Madagascar and South Africa. The 1995 AFRAN Directory of Nephrology mentions only 4 of these in relation to nephrolgists and dialysis /transplantation centres. The zone is beset with poor finances and, as a consequence of civil uprising and struggling economics the practise of "high tech" medicine is limited. Direct contact with the embassies in attempting to define the details of dialysis facilities was unhelpful. A medline search of published material from this zone was also undertaken. It is clear that the strength of Nephrology lies in the south. There are more publications, on matters Nephrology and more dialysis units with support transplantation than anywhere else in the zone. In addition the private sector has built up a strong presence. This has arisen partly as a consequence of budgetery restrictions imposed by the department of health. The ability to easily communicate with other nephrologists outside of South Africa is exceedingly difficult. If AFRAN is to play any role in facilitating better "renal treatment" then channels of reliable communication must be established so that we can speak to each other in Africa. If not then AFRAN will become an organization with no future.

The role of leukocytes in glomerular injury. *Adu D., Dept. of Nephrology, The Queen Elizabeth Hospital, Birmingham.* A key feature of antibody mediated glomerulonephritis is the intra-glomerular accumulation of leukocytes. This is seen in post-infectious glomerulonephritis

and also in auto-immune glomerulonephritis such as anti-GBM disease, lupus and vasculitis. The migration of leukocytes from the lumen of blood vessels is dependent on a series of activation steps that include rolling, adhesion, diapedesis and migration. The first step is dependent on selectins (P-Selectin and E-Selectin) on endothelium and enables the leukocytes to sample chemoattractants (chemokines) bound to proteoglycans on the endothelial cells. Chemokines lead to activation and upregulation of leukocyte integrin receptors which bind adhesion molecules ICAM-1 and VCAM-1 on endothelium. Subsequently leukocytes migrate across the endothelium up a chemoattractant gradient. On in vitro human glomerular organ culture, glomerular endothelium can be induced by cytokines to express E-Selection and VCAM-1 and to increase the expression of ICAM-1. In the renal biopsies of patients with active glomerulonephritis there is an increase in glomerular endothelial expression of ICAM-1 and new expression of VCAM-1. In vitro studies show that these adhesion molecules are able to bind human leukocytes. Recent studies show that there is an increased expression of chemokines in the glomeruli of patients with a glomerulonephritis. These chemokines which include MCP-1, MIP-α/β, RANTES and IL-8 attract different subsets of leukocytes and thereby confer specificity to leukocyte recruitment. Once within glomeruli, activated leukocytes release reactive oxygen products and proteases which lead to tissue injury and in addition produce cytokines including IL-1 and IL-8 which in themselves augment further leukocyte recruitment and activation. A clear understanding of the mechanism of leukocyte recruitment into glomeruli will lead to the development of novel therapies.

Lupus nephritis (LN). *Nctkong, Department of Medicine, National University of Malaysia, Kuala Lumpur.* Renal involvement occurs in about 50–80% of patients with SLE. Immunopathogenetic studies are now focussed on dysregulation of apoptosis which may lead to quantitative or qualitative changes in the release of nucleosomes which may be the major autoantigen rather than the poorly immunogenic dsDNA. Nucleosome antibodies coupled with nucleosomes are demonstrated to have a high affinity for the GBM resulting in LN. LN presents in many ways and variable histological patterns or combinations thereof (WHO classification) are found on renal biopsy. The histopathologic class of LN is highly correlated with prognosis and an aggressive therapeutic approach to severe LN has resulted in a markedly improved renal survival. The goals of treatment of severe active LN encompasses remission induction, remission consolidation and remission maintenance in the short term PLUS prevention of progressive renal failure and minimization of drug toxicity in the long term. To this end, low dose steroid-cytotoxic combinations are superior to high dose steroids alone. IV cyclophosphamide is now widely employed although optimal dosing and duration of therapy remain controversial. Azathioprine may be preferable for chronic therapy. Plasmapheresis is a useful adjunct for refractory disease. The role of other therapeutic modalities such as cyclosporine A, methotrexate, IV immunoglobulins, FK506, lefluomide and total lymphoid irradiation remain to be defined. However, prospects for specific immunotherapies are novel and promising and include monoclonal antibodies, soluble receptors and receptor ligand blockers amongst others. Ultimately, perhaps gene replacement therapy.

Why has improvement in hypertension treatment not reduced the incidence of end-stage renal disease? *Yackoob K. Seedat, Department of Medicine, University of Natal, Durban, South Africa.* Hypertension is an important cause of End-Stage Renal Disease (ESRD) in the USA and in Sub-Saharan Africa. Antihypertensive therapy has led to a substantial decrease in incidence of stroke (42%) and to a lesser extent of myocardial infarction (16%), yet there has been an increase in hypertension related (ESRD). In 1991, about 190,000 persons in the USA either underwent dialysis or received a transplant for ESRD. Hypertension was found to be the underlying cause in 29% of these patients, second only to diabetes mellitus (36%). Both in the USA and South Africa hypertension was found to be the most common cause of ESRD, but it is not clear whether this is related to the higher incidence and severity of hypertension in blacks. Moreover BP control in black patients does not necessarily lead to improved renal function. These findings suggest that factors other than BP elevation participate in the progression of nephroclerosis. They include misdiagnosis, black race (socio-economic status, physiologic differences, severity of hypertension), BP control (adequate control of BP and type of antihypertensive therapy), and possibly other factors like genetics. The lack of reduction in ESRD may be that currently accepted standards for BP control are not adequate and that different antihypertensive agents affect glomerular haemodynamics in different ways. These factors need an indepth analysis to improve the important public health issue of increasing morbidity and mortality from ESRD.

Acute renal failure: New perspectives on pathophysiology, prevention and treatment. *Charles L. Edelstein and Robert W. Schrier, Department of Medicine, University of Colorado School of Medicine, Denver, Colorado, USA.* Acute renal failure (ARF) is a very common renal disease affecting about 5% of all hospitalized patients. ARF still carries a very high mortality of more than 50% and there has been no significant change in mortality over the last four decades. In the last decade several new and important pathophysiological mechanisms that underlie the renal dysfunction have been discovered. These pathophysiological mechanisms include the role of 1) both calcium and calcium-dependent enzymes like calpain, 2) nitric oxide, 3) oxidant stress, 4) loss of polarity of the tubular cell, 5) tubular obstruction and arginine-glycine-aspartic acid (RGD) peptides, 6) neutrophils, 7) intercellular adhesion molecules (ICAM), 8) vascular factors e.g. loss of renal blood flow autoregulation, 9) atrial natriuretic peptide and 10) growth factors. A better understanding of tubular and vascular mechanisms has led to therapeutic studies in animals and clinical trials in humans. For example, the systemic administration of cyclic RGD peptides in the rat ameliorates ischemic ARF by preventing tubular obstruction. Also, in vivo targeting of inducible nitric oxide synthase with antisense oligodeoxynucleotides protects the rat kidney against ischemia. In human studies, the use of biocompatible dialysis membranes enhances recovery of renal function and patient survival in ARF. In this lecture, the pathophysiology of ARF will be correlated with the rationale for both current and future therapies.

Anaemia of chronic renal disease and pathogenesis and management, with reference to the use of erythropoietin and intravenous iron. *Nathan W. Levin, Renal Research Institute, New York* The investigation of the anemia of chronic renal failure has hardly changed for many years. However, the availability now of Epoetin and intravenous iron has transformed the management of anemia with consequent well-documented evidence of physical and psychosocial benefit. Current issues for debate include the methods for prescribing Epoetin, level of the target hematocrit (hemoglobin), whether oral iron is needed (in hemodialysis), how to assess iron status, concerns about the rate of administration of IV iron and safe levels for body iron, and the comparison of the relative efficacy of subcutaneous versus intravenous iron. The concept of functional iron deficiency requires that intravenous iron be given in substanially larger amounts and therefore to a higher level of transferrin saturation and of ferritin. The risk of this approach is still under investigation with the role of reactive iron species in promoting oxidative damage being of particular interest. The benefits are controversial. The use of EPO in hospitalized patients with infective or posttraumatic problems is not clarified. The interim somewhat negative results of the "Normalization of Hematocrit Study" in dialysis patients with heart failure (as opposed to other patients) require satisfactory explanation. The improvement in survival in ESRD patients in whom the hematocrit reaches to 32-36 is well documented. Cardiovascular correlates are of interest. The just issued (U.S.) National Kidney Foundation-Dialysis Outcomes Quality Initiative Practice Guideline on anemia deal with most of the above topics with supporting evidence and rationales.

Economy in renal health care in Africa. *Aziz El Matri, Faculty of Medicine, Tunis.* Of the hundred of thousands of people in the world with end stage renal disease (ESRD) about 75% live in the developing world and about 12% in Africa. In Africa, the average Gross National Product (GNP) per capita is US $600, i.e. 7 times lower than the average GNP. Twenty two out of 53 countries have a GNP per capita lower than 400 US $ and are classified among the 40 poorest countries in the world. Some countries have even a negative economic growth.

It is proven that there is usually a correlation between the yearly per capita expenditure in a country and the level of health care. In the continent, and mainly in sub-saharian Africa, there are many endemic infectious and parasitic diseases and health indicators are generally poor as for example the ratio of doctors/10000 population is ranging between 0.9 and 1.5 and yearly expenditures per capita for health care is ranging between 9 and 28 US $. As for renal diseases, they are particularly common and are associated with very high morbidity and mortality. Acute renal failure remains fairly common. Its incidence, though not precisely defined is ranging between 100 and 150 per million population per year. The reported incidence of ESRD ranges between 89 and 192 per million population. Haemodialysis is available for management of acute renal failure in most of the major hospitals of the capital cities. The number of patients treated by regular dialysis in Africa cannot be precisely defined owing to the scarcity of reliable registries. On the other hand availability of dialysis services in certain countries does not reflect the accessibility. Renal transplantation is performed mainly in North Africa, South Africa, Zimbabwe and Kenya. So the countries of sub-saharian Africa represent what is undoubtly the worst cases and medical staff and decision makers should act together to improve it. In Africa where the population is 670 million and where the incidence of ESRD is at least 100 per million population, there would be a prevalent population of some 250 000 patients after 5 years. If the cost of haemodialysis for 1 patient for 1 year is US $12000 as reported by North African studies (in USA US $25000 to 30,000), the total expenditure for haemodialysis would be US $2000 million per year. It is high when you know that in certain countries the cost of keeping one patient alive on dialysis exceeds the average GNP generated by 10 citizens. But it is necessary to set up national programmes of treatment of ESRD. It may be that the changes in political climate we are witnessing now will lead to a situation where reduction in domestic and political turmoil will allow these nations to effectively address domestic social and health problems. So Nephrology and mainly ESRD must have a place in the hierarchy of health priorities and it would be reasonable to divert funds to their treatments. Nationalization of the issues of nephrologic diseases and renal failure management should be a pressing target of doctors in Africa. It cannot be over emphasized that very little indeed can be achieved, unless opinion leaders, decision makers, fund raising organizations and public information media are recruited into the same boat. According to the African tradition of solidarity the healthy majority of the people should support the sick minority.

Renal disease/failure in Egypt. *Maher F. Ramzy, M.D.—F.A.C.P, Professor of Medicine & Director of Nephrology Centre—Cairo University.* **Renal disease/failure**: The exact incidence of renal failure is unknown. Analysis of health certificates showed that 192/M die with renal failure every year. (Barsoum et al, 74) Acute renal failure: The incidence seems to be decreasing. Major surgery is associated with high risk of A.R.F. Cresentic G.N. (Panci-immune) seems to be increasing. Drugs, hydrocarbons and other environmental factors has to be considered. Chronic renal disease/failure: Despite the lack of registry, a lot of data point to increasing of H.T.N. renal disease, diabetic nephropathy and chronic interstitial nephritis. The latter probably accounts for most cases of ESRD of unknown aetiology. This could be related to enviromental pollution, industrial toxins and drug misuse. In the dialysis pool, the percentage of diabetics is increasing from 5.2% to 8.4% (El Sharkawy, 93). One in four Egyptian is or will become H.T.N. (Ibrahim M, 95) this points to possible magnitude of related renal disease. In adults the predominant pattern of primary G.N. is mesangiocapillary G.N. (Ramzy et al, 90); however G.N. as cause of ESRD dropped from 30.4% to 20.2% (El Sharkawy M, 96). HCV and other infections are major causative factors. In nephrotic children, increased incidence of focal segmental sclerosis (up to 25%) is observed (Sohh et al, 90). **Treatment modalities**: Dialysis: One hundred and thirty four dialysis units are recognized, a lot are not registered yet. The pool of patients on chronic dialysis is continuously expanding during the last three decades; it is about 245/M of general population (Ministry of Health data, 1996). Most pts. are on ch. HDx, few on intermittent PD and occasional cases are treated by C.A.P.D. The majority of pt. are inadequately dialyzed; the 1st year mortality on dialysis is >50%, the annual mortality thereafter is about 35% (El Saeed et al, 95). Renal Transplantation: Started at Mansoura in 1983. The actual number is unknown, probably six/M (360/y). Results of rental transplantation are comparable to international standards. Most donors are live unrelated. A law for cadaveric donation is still absent. Strict rules are now applied to control the Egyptian kidney market. **Funding**: Eighty percent of chronic dialysis pts. are sponsored by Ministry of public health.

Nephrology in Tunisia. *Ben Abdallah T., Kheder A., Ben Moussa F., El Younsi F., Abderrahim E., Ben Hamida F., Goucha R., El Matri A., Ben Maiz H, Service de Nephrologie et de Medecine interne (Pr. Ben Maiz H.) Charles Nicolle University Hospital. Tunis—Tunisia.* Nephrologic care has been available in Tunisia since 1962 but it has developed particularly since 1968 when a comprehensive hemodialysis programme was set up in Charles Nicolle Hospital in Tunis. Renal Biopsy has been performed since 1967. Until December 1996, 4492 biopsies have been done. An intermittent peritoneal dialysis programme has been active since 1982 and has been completed by CAPD in 1983. Renal transplantation has been performed abroad since 1971 but a national programme was started in June 1986. Today Nephrologic care including hemodialysis and renal transplantation is available in Tunisia at 4 University Hospitals for adults and one nephrologic pediatric unit in Tunis. Until December 1996 there are 72 Hemodialysis centers (15 Public and 57 private). With 2849 patients (313 pMp with a progression in 1996 of 32.5 pMp): 2796 were treated by hemodialysis, 43 by CAPD and 9 by IPD. About 210 renal transplantation (RT) were performed in Tunisia among them 171 in Charles Nicolle Hospital. Distribution of main renal diseases are: glomerulonephritis 20.4%, pyelonehritis 13%, vascular disease 17.5%, diabetic nephropathy 11.8%, hereditary nephropathy 6.4% and unkown 30.9%. Plasmapherisis has been available at Charles Nicolle Hospital since 1983. The incidence of acute renal failure (ARF) in Tunisia is about 15 pMp: 120 cases per year. The main aetiologies of ARF are: Functional ARF 22 to 47%, Acute tubular necrosis 13 to 37%, among them 17% were obstetrical and 68% toxic, Urological ARF 10%, Acute glomerulonephritis 19% and Vascular ARF 1%. Expenses of renal replacement therapy are on the charge of Minestry of Health or social security funds. Hemodialysis costs about US Dollars 18,000, CAPD 15,000/patient/year and RT 19,000 the first year. The Tunisian Society of Nephrology was created in November 1983. It organizes 2 to 3 scientific meetings per year.

Clinical course and management of viral hepatitis in renal transplant recipients. *Venkateswara K. Rao, MD, Professor of Medicine, University of Minnesota Medical School, Minneapolis, MN.* Approximately 15% of renal transplant recipients develop chronic hepatic dysfunction. Infection with hepatitis B and C viruses is the predominant cause of chronic liver disease. Because of the immunosuppressed state, intra hepatic viral replication continues to occur which leads to post necrotic cirrhosis and hepatic failure over a period of 10 to 15 years. Hepatic failure with or without super imposed sepsis is the leading cause of death in renal transplant recipients surviving into the second decade. These patients remain asymptomatic despite ongoing histological progression. Liver enzymes are only modestly elevated and fluctuating levels from normal to abnormal are quite common. There is no correlation between level of hepatic enzymes and histologic severity of liver disease. Initial morphology has a predictive value in terms of subsequent histological and clinical progression and patient mortality. Older age, female sex and morphologic diagnosis of chronic active hepatitis are associated with progression to cirrhosis in the post transplant period. Hepatitis B patients have significantly more severe morphologic forms of liver disease and have higher incidence of death from hepatic failure compared to those with hepatitis C. With improved surveillance measures and pre transplant vaccination, the incidence of hepatitis B has declined steadily in the United States. A third of renal transplant patients now have hepatitis C infection, of which another 1/3 develop clinical disease. It is our policy to obtain a liver biopsy in HCV infected patients if they have clinical disease or have high viral RNA titer. Those with histologic evidence of chronic active hepatitis are treated with alpha interferon. Contraindications are mental depression, neutropenia, frequent acute rejections, significant hepatic fibrosis or cirrhosis in the biopsy and clinical hepatic failure. Biochemical and histologic improvement was observed in several of our patients who were treated

with interferon. The patients, however, need close follow up to monitor the side effects, particularly, renal function. Most patients require long term therapy to control the disease, provided they are able to tolerate the medication. Every effort should be made to prevent the infection by screening the blood and organs for HCV along with other standard surveillance measures during the pre and post transplant period.

Strategies for the treatment of glomerulonephritis. *Adu D., Dept. of Nephrology, The Queen Elizabeth Hospital, Birmingham.* Our current treatment of glomerulonephritis is based on the concept that most of these disorders are immune mediated. Our lack of understanding of the precise mechanisms leading to this immune activation means that our therapies are based on overall suppression of the immune system. Nevertheless current treatment strategies are associated with improved survival and reduced drug toxicity. This is likely in part to be due to better treatment of the consequences of glomerulonephritis e.g., hypertension and hyperlipidaemia. In some types of infection associated glomerulonephritis, e.g. that due to endocarditis and Hepatitis C eradication of the infection (and hence of antigen) leads to resolution of the glomerulonephritis. This strategy is not effective in glomerulonephritis due to malaria and schistsomiasis probably because these infections trigger an autoimmune response which is then responsible for the glomerulonephritis. Steroids and Cyclophosphamide are effective in inducing remission in minimal change nephropathy and to a lesser extent in FSGS. Here there is evidence of a circulating proteinuria inducing factor, possibly produced by lymphocytes. Of interest these disorders recur after transplantation and remission can be induced by plasma exchange which removes the circulating factor. In the classical autoimmune disorders anti-GBM nephritis and lupus nephritis, there is the glomerular deposition/assembly of antibodies and complement. There is clear evidence that steroids and Cyclophosphamide are effective in treatment. By contrast in idiopathic membranous nephropathy and MCGN in which there are also glomerular immune deposits, steroids and immunosuppressants are either ineffective or of dubious efficacy. Their reasons for this are not clear. In Wegener's granulomatosis and microscopic polyarteritis antibodies directed against neutrophil cytoplamsic activate neutrophils and may thereby lead to glomerular injury. Here treatment with Cyclophosphamide and steroids are effective in inducing remission of both the glomerulonephritis and vasculitis. We need to develop treatment strategies that focus on the likeihood that the drugs needed to induce remission may be different from those needed to consolidate remission and to prevent relapse. This is already possible when there is a good immunological marker, e.g anti-GBM antibodies and ANCA that correlated with disease activity.

Hepatitis B virus-associated nephropathy in black South African children. *Bhimma R., Coovadia H.M., Adhikari M., Department of Paediatrics and Child Health, University of Natal, Durban, South Africa.* Hepatitis B virus (HBV) is the prime cause of membranous nephrotic syndrome (NS) in South African children but the spectrum of glomerular injury has not been adequately documented in black children. One hundred and thirty three children with NS in whom HBV infection was detected are included in this study. In 70 patients the histological type was membranous (HBMN); of whom 46 were followed up for a mean of 3.4 years (range 1–11). Spontaneous elimination of both HbsAg and HbeAg occurred in 10 (22%) patients while 16 (35%) cleared HbeAg alone. Co-existing liver disease occurred in 3 (4.3%) and hypocomplementaemia (C3, C4) in 47.1 and 11.4% of children respectively. 65 (92.9%) patients had normal renal function; 1 (1.4%) impaired renal function; 3 (4.3%) chronic renal insufficiency and 1 (1.4%) end stage renal disease at last hospital visit. 12 of these patients were in remission and all had become free of HbeAg. HBMN was clinically indistinguishable from 24 children with idiopathic membranous nephropathy (IMN) although serum triglycerides, complement components (C3, C4, Factor B) and serum globlulins (alpha 1, alpha 2, beta) were different. There were 23 patients with histological lesions other than HBMN, of whom 6 (26%) had diffuse mesangial proliferation, 5 (22%) focal segmental glomerulosclerosis, 5 (22%) membranoproliferative glomerulonephritis, 4 (17%) focal mesangial proliferation and 3 (13%) had intermediate changes (unclassified). Forty patients were unbiopsied and had clinical, biochemical and serological findings similar to those with HBMN and the other histological types. This report delineates the natural history of HBV infection in black South African children with membranous nephropathy, although other histological patterns may also be seen.

Acquired hair and skin fairness in hemodialisis patients. *M. Ben Hmida, N. Abbes, K. Kammoun, S. Bouacida, K. Charfeddine, H. Turki, J. Hachicha, P. Reygagne, D. Rabier, A. Zahaf, A. Jarraya, Dpts. of Nephrology—Dermatology. CHU—Hédi Chaker—3029 Sfax.* With recent advances in medicine, uremic patients are living longer with an improving quality of life. Several skin diseases have been reported in patients with chronic renal failure, and the opportunity has been offered to elucidate newer cutaneous abnormalities among patients undergoing long-term hemodialysis. Hyperpigmentation was the most prevalent cutaneous abnormality observed in these patients, but hypopigmentation remains an exceptional event. We report here a series of 6 maintenance hemodialysis patients (5 M + 1 F) with an acquired hair and skin fairness. Mean age was 42 years (range 23–56). Mean duration of hemodialysis therapy was 52 months (range 10–96). The determination of the hair aminoacid composition was carried out. A polarized light micrograph and scanning electron micrography of head hair were performed. This clinical feature seems to be similar to the 'dilution' of hair color observed in phenylketonuria. A low plasma tyrosine level with normal plasma phenylalanine concentration corroborates this diagnosis in these cases. This apparent resemblance would suggest an acquired impairment of phenylalanine hydroxylase activity in uremic and hemodialyis patients. The mechanism of this impairment will be discussed.

Congestive heart failure in elderly hemodialysis patients. *Pedro Leão Neves, Idalécio Bernardo, Centros de Diálise de Faro e Portimão. FMC. Algarve. Portugal.* Cardiovascular disease is the main cause of death in chronic hemodialysis patients (CHD). Congestive heart failure (CHF) either preceding or appearing after the initiation of CHD is also associated with increased mortality. In this study, carried out in the centres of Faro and Portimão, we included all patients aged over 65 y (N = 67, f = 33, m = 34, mean age = 72.6 y, mean time on CHD = 51.3 months). We looked for the presence of CHF after the initiation of CHD and we divided the population in two groups: patients with CHF (G–I = 15) and without CHF (G–II = 52). We compared both groups respecting several biological, laboratorial and echocardiographic parameters. There were no differences between groups respecting age, sex distribution, time of hypertension preceding CHD, time on CHD, blood pressure levels, interdialytic weight gain and for the presence of ischaemic heart disease. The body mass index (BMI) (19.8 vs 21.9, $p = 0.066$) and the plasma creatinine level (8.1 vs 9.4 mg/dl, $p = 0.018$) were lower in G–I. The other laboratorial parameters analysed were not different between the groups. The G–I showed a higher left ventricular mass index (LVMI) (209 vs 147 g/m^2, $p = 0.001$) and a lower fractional shortening (30.2 vs 38.6%, $p = 0.001$). The left ventricular diastolic diameter (LVDD) was higher in G–I (5.5 vs 4.91 cm, $p = 0.001$), but both the left ventricular posterior wall and the interventricular septum thickness were similar. In conclusion, the results showed that our elderly hemodialysis patients with CHF have: 1—lower BMI as well as lower creatinine levels (probably with the same meaning) 2—lower fractional shortening and a higher LVMI due to an increased LVDD.

Soluble interleukin-2 receptors in renal failure and early post-transplantation. *Mohamed Hani Hafez and Inas Raafat, Departments of Nephrology & Clinical Pathology, Cairo University.* Soluble Interleukin-2 receptors (sIL-2R) released during T-lymphocyte activation were Quantified by ELISA. Forty subjects were included and divided into control, acute rejection, stable transplant, hemodialysis and uremic conservation groups. In 16 renal failure patients mean sIL-2R was significantly higher than controls, with higher values reported with hemodialysis ($p < 0.05$). This could be caused by non-dialyzable nature of sIL-2R molecule and altered IL-2 dependent pathway. There was no significant difference between those treated with erythropoietin and others. SIL-2R did not correlate with hemoglobin but correlated

positively with serum creatinine. Transplanted patients were evaluated to exclude cyclosporin toxicity, acute tubular necrosis or concomitant infection. Among transplantation cases, eight showed criteria of acute rejection and eight had stable graft function. Pre- then post-transplantation samples were taken within two weeks after surgery. Pre-operative MHC and sIL-2R, intraoperative ischemia time were not significantly different and therefore did not predict allograft loss. Patients with stable graft, when compared to their initial levels showed significant drop of sIL-2R after transplantation ($p < 0.001$), following pattern of serum creatinine concentration ($r = 0.54$, $p < 0.05$). In patients with graft rejection, significant rise in sIL-2R ($p < 0.02$) was obtained in comparison to their pre-transplant values. Serum sIL-2R and creatinine increased with positive correlation ($r^* = 0.46$, $p < 0.1$). Serum sIL-2R is elevated at diagnosis of uremic syndrome and remains so during hemodialysis treatment. It is only after successful renal transplantation that levels decrease, thus it is an additional non-invasive test diagnosing acute rejection. SIL-2R follow-up could have more implications with the use of anti-CD25 in clinical transplantation.

Long term follow-up of kidney donors. *S. Naicker, M. Kirsten, A. Sookoo, I. Holmes, A. Muranda, A.A. Haffejee Renal Unit, Addington Hospital and Depts. of Medicine and Surgery, University of Natal, Durban, South Africa.* There is a worldwide shortage of organ donors. The problem is more pronounced in our province of Kwa Zulu/Natal where the rate of cadaver organ donation is very low and therefore both patients and nephrology staff are under pressure to seek living donors. We studied 135 living donors who underwent nephrectomy in a 10 year period: 85 females and 50 males; 78 (57.8%) were of Indian origin, 33 (24.4%) Black, 15 (11.1%) White and 9 (6.7%) of mixed race groups. The majority of donors (57%) were siblings, while 14.8% were parents, 6.7% children, 17.8% spousal and 3.7% were cousins. The mean age of the donors was 34.2 years (range 21–56 years). Donors were hospitalised for a mean of 6.1 days (range 3–15). Post-operative complications noted were left lower lobe atelectasis and chest infection in 11.1%, other infection in 5.2%, pneumothorax in 2.2%, ileus in 2 pts, depression in 1 pt and prolonged pain at the site of surgery in 11.1%. Increase in proteinuria was noted in 3 pts (0.4 grams daily in one pt at 9 years and 0.26 grams and 0.66 grams in 2 pts at 2 years). Blood pressure recordings were virtually unchanged from pre-nephrectomy levels. This study suggests that unilateral nephrectomy in normal individuals is associated with few major adverse effects and living donor kidney transplantation is a viable option.

The 4th decade of renal transplantation: Where have we come? Whither are we going? *L.P. Margolius & J.R. Botha, University of Witwatersrand, South Africa.* Since the advent of renal transplantation on 21st August 1966, 1786 renal transplants have been performed in 1483 patients at Johannesburg Hospital in South Africa. During this period 1222 patients have received primary cadaver grafts, 303 patients subsequent grafts and 249 patients related living donor grafts. 12 patients have received unrelated living donor grafts (from spouse or close friend). The aim of this paper is to report the results of the various groups, to elucidate on some points and to attempt to explain discrepancies between our results and the literature.

Actuarial survival	1 year		5 years		10 years		15 years		20 years	
	Pt	Gr	Pt	Gr	Pt	Gr	Pt	Gr	Pt	Gr
All patients	83	61	73	47	58	32	47	22	34	14
RLD	92	78	89	67	79	48	71	36		
CD1 all	82	59	69	44	53	29	40	20	30	20
CD2 all	82	52	77	41	60	27	55	19		
CD1 CYA	85	63	71	46	53	32				
CD1 no CYA	76	53	64	40	51	26				
CD1 black	84	58	70	37	36	17				
CD1 white	92	78	91	69	81	51				

Abbreviations are: Pt, patient; Gr, graft.

Optimal patient and graft survival appears to depend on close HLA matching, optimal crossmatch techniques use of Cyclosporine based therapies and short ischaemic time.

Renal function and sodium handling in patients with portal vein thrombosis compared to normal controls. *Rayner B.L., Voigt M., Robson S., Kirsch R., Renal Unit and MRC Liver Research Centre, University of Cape Town.* Patients with cirrhosis of the liver have impaired ability to excrete salt and water, and may also be prone to develop renal failure due to the hepatorenal syndrome. The precise mechanisms behind this are largely unknown but porto-systemic shunting may be an important contributing mechanism. Our aim was to study renal function and sodium handling in patients with portal vein thrombosis without liver disease (PVT) compared to normal controls to determine the role (if any) of porto-systemic shunting. Nine patients with proven PVT and 9 sex and age matched controls were studied. All underwent standard measurements of GFR and renal blood flow using inulin and PAH acid clearances, and sodium excretion, before, during and after an intravenous saline challenge. Renin and aldosterone levels were also measured before and after. At baseline there was no difference in inulin clearance, PAH clearance, fractional excretion of sodium, renin or aldosterone. During and after the saline infusion there was a drop in the PAH clearance and a concomitant increase in the fractional excretion of sodium. Aldosterone and renin levels fell after the infusion. There was no difference between the PVT and control groups. In conclusion renal function and sodium handling was very similar between controls and patients with PVT. It is, therefore, unlikely that portosystemic plays a significant role in the genesis of the hepatorenal syndrome or the impaired ability to excrete sodium in patients with liver cirrhosis.

Urinary tissue Kallikrein excretion in severe hypertension of pregnancy: Decrease and correlation with uric acid and renal impairment. *S.M. Khedun, T. Naicker, J. Moodley, S. Naidoo, K.D. Bhoola, Department of Pharmacology EM Unit and MRC Pregnancy Hypertension Research Unit, Natal Medical School, Durban.* The aim of this study was to establish whether urinary tissue kallikrein (uTK) excretion was increased in severe hypertension of pregnancy and if these levels correlated with other laboratory markers of severity of this complication. Random untimed urine samples were collected at 28 weeks of gestation and near at the time of delivery from all patients; 66 had severe hypertension of pregnancy and 66 were normotensive pregnant women. Urine specimens were analyzed for uTK using a chromogenic substrate S2266. uTK levels were decreased in severe hypertension in pregnancy compared with normotensive pregnant women (1.55 ± 0.95 vs 3.02 ± 2.5 ng TK/μg protein; $p < 0.0001$). uTK excretion correlated positively with uric acid ($r = 0.72$; $p < 0.0001$) and serum creatinine levels ($r = 0.84$; $p < 0.0001$). There was a significant difference in uTK excretion between 28 weeks of gestation and at near delivery in the severe group (1.94 ± 0.65 vs 1.21 ± 0.25 ng TK/μg protein; $p < 0.0001$). This study shows that uTK excretion is decreased in severe hypertension of pregnancy and correlates closely with uric acid and serum creatinine the measure of renal function.

Plasma vasoactive peptides in renal disorders. *P. Gathiram, S. Naicker, A. Nadar, J. Duursma, Dept. of Physiology, University of Durban-Westville.* The mechanism of occurrence of hypertension in renal disorders is postulated to be multifactorial, including salt and water retention, imbalance between vasoconstrictor and vasodilator substances. In this study the role of plasma endothelin-1 (ET-1), atrial natriuretic peptide (ANP) and renin-angiotensin system (renin activity) was assessed in haemodialysis (HD), continuous ambulatory peritoneal dialysis (CAPD), and renal transplant (TP) patients and compared with normal controls. Plasma ET-1 was measured using an EIA kit (Wako Chemicals) and ANP using an RIA kit (Peninsula Lab.) after extraction of the samples using Sep-Pak C18 cartridges and renin activity by RIA (Biodata). Compared to controls there was a significant elevation in plasma ET-1 (0.004) in all groups while plasma ANP ($p < 0.006$) and renin activity ($p < 0.05$) were significantly elevated in both dialysis groups.

Group	ET-1 (*pg/ml*)	ANP (*pg/ml*)	Renin (*ng/ml/h*)
HD	3.31 ± 1.39	87.2 ± 45.8	6.42 ± 7.5
CAPD	2.96 ± 0.95	54.4 ± 17.6	6.01 ± 5.8
TP	3.1 ± 0.32	33.8 ± 12.3	3.42 ± 2.18
Control	1.57 ± 0.13	24.3 ± 2.91	1.42 ± 0.86

A positive correlation was found between plasma ET-1 levels and ANP in CAPD (r = 0.7) and HD (r = 0.89) patients. Our results support the clinical observation that the largest changes in volume and blood pressure occur in dialysis patients with concomitant changes in pressor substances.

Insulin-like Growth Factor-1 (IGF-1) and uraemic cardiomyopathy. *E.C. Muchaneta-Kubara, T.S. Johnson, A.M. El Nahas, Sheffield Kidney Institute, Northern General Hospital Trust, Sheffield, UK.* The mortality of patients with end stage renal failure is related to the severity of left ventricular hypertrophy (LVH). Since IGF-I has been put forward as a myocardial growth factor, we have studied the changes in myocardial IGF-1 during the course of experimental uraemia in subtotally nephrectomized (SNx) rats. Controls included sham-operated Wistars and spontaneously hypertensive rats (SHR). Animals (n = 6) were sacrificed on days 7, 30, 90, and 120 after SNx. Extractable (eIGF-1) and immunostainable (iIGF-I) myocardial IGF-I were determined by radioimmunoassay and immunohistochemistry respectively. IGF-I gene expression was studied by Northern blot analysis of IGF-I mRNA. Uraemic cardiomegaly was due primarily to hypertrophy as shown by a significant increase in myocardium protein content (71.7 ± 14 mg/heart) compared with sham (39.2 ± 5 mg/heart) $p < 0.001$. Histologically, perivascular and inter-myocytic fibrosis was detectable from day 90 onward. A biphasic increase in myocardial eIGF-I was noted with an early peak on day 7: 235 ± 32.9 ng/heart, compared to sham (128.6 ± 14.6ng/heart) $p < 0.001$ and another peak on days 90-120, with no changes detected in SHRs. The iIGF-I was raised in uraemic myocytes throughout the study. A positive correlation was noted between eIGF-I and IGF-I mRNA ($r = 0.551$) $p < 0.01$. Progressive uraemic cardiomyopathy is associated with elevated myocardial IGF-I content and expression.

Diagnosis of active CMV infection in renal transplant recipients by CMV antigenaemia. *Z. Noveljic, A.A. Walele, E.J. van Rensburg and M.R. Moosa, Dept. of Virology and Medicine, University of Stellenbosch, Cape.* Active CMV infection in an important complication early post-transplantation. It is difficult to diagnose as current diagnostic methods have major drawbacks. The aim of the study was to establish a diagnostic value of the CMV antigenemia (CMV Ag) assay. During the period July 1996 and April 1997 all new renal transplant recipients were monitored for active CMV infection during the first four months post-transplantation. Weekly sampling of whole blood peripheral leukocytes was assessed for CMV by conventional virus culture, detection of early antigen in rapid culture (shell vial culture) and direct detection of the CMV lower matrix antigen pp65 (CMV Ag). A positive CMV Ag was defined as >10 per 50000 WBC. Active CMV infection was defined as CMV Ag > 30 per 50000 WBC or a positive virus culture. Clinical manifestations were retrospectively correlated with the laboratory findings. Thirteen consecutive renal transplant patients were studied. CMV Ag was positive in eleven (85%). CMV Ag > 30 per 50000 WBC was detected in four patients with two confirmed on conventional virus cultures. CMV was clinically suspected in three of them. CMV Ag 10–29 per 50000 WBC was detected in seven patients and all were negative on conventional virus cultures. CMV was clinically suspected in three of them. The detection of CMV by rapid culture was negative in all the patients. Expressed quantitatively CMV Ag could discriminate between CMV infection and CMV disease. A high CMV Ag result suggestive of CMV disease was confirmed by conventional viral cultures.

Urinary leakage post renal transplantation. *M. Jemni*, M. Hajri*, S. Karray*, S. Friaaa*, M. Chebil*, T.B. Abdalah**, H.B. Maiz**, M. Ayed*, *Department of Urology; **Department of Nephrology, Hôpital Charles Nicolle, Tunis.* From 1986 to 1996, 160 patients underwent kidney transplantation. 95% of transplantations were from a relative alive donor. We had 11 ureteral fistulas (6.8%). Fistula incidence in Leadbetter-Politano was 5.8% and 8.1% in Lich-Gregoir reimplantation ($p = 0.8$). 8 patients were treated surgically and 3 by percutaneous procedure. Intra-operative findings consisted in an extended necrosis of the ureter in 4 cases, distal ureteral, necrosis in 1 case, ureterovesical leakage in 2 cases and a limited pyelic fistula in 1 case. Surgical procedure consisted in pyelo-ureteral anastomosis with native ureter in 4 cases, a new ureteral bladder implantation in 3 cases and one patient had a simple suture of a limited pyelic fistula. All patients had their fistulas closed, 7 patients had no alteration of renal function but only one patient had altered his renal function. The patients treated percutaneously had nephrostomy and double J ureteral stent. One patient had a total recovery, one patient had ureteral stenosis which was successfully managed surgically and the last dead with sepsis. More than 50% of ureteral fistulas are due to ureteral necrosis. We insiste on the importance of preserving ureteral vascularisation. During renal harvesting. Surgical management for ureteral fistulas provides the best results.

Acquired cystic kidney disease in hemodialysis patients. *Hachim K., Benghanem Gharbi M., Fatihi E., Zahiri K, Ramdani B., Zaïd D, Service de néphrologie, CHU Ibn Rochd, Casablanca, Morroco.* This study carried out over 62 patients undergoing long term dialysis. We have discovered an incident of acquired cystic kidney disease reaching 33%. Sex influence has been evident: 61.9% among men and 38.09% among women have this pathology. Also, hemodialysis period has an influence on its emergence. It comes in average to 85 ± 47 months to patients with acquired cystic kidney disease, against 49.5 ± 37.5 to patients without. According to age and causes of renal failure in dialysis patients, they have no influence. Most of our patients are anuric (57.1%) and badly purified. They are asymptomatique in 33.33% of the cases, presenting flanc pain in 57.1%, hematuric in 6.5%, and urinary infection in 19.04%. The hematocrit ratio depends mainly on cystic proliferation and not on the presence of the cyst. That ratio is about 22 ± 7 in Grade 1 patients according to Goldsmith Scale; in the same way the Grade 3 patients have the ratio in 29.6 ± 6.3. The cystic haemorrhage has been the only complication, observed in 4.7% of the cases. Renal cancer has never been detected in any patient.

Chronic hemodialysis for children first Moroccan experience. *Fatihi E., Benghanem G.M., Hachim K., Zahiri K., Ramdani B., Zaid D., Service de néphrologie-hémodialyse CHU Ibn Rochd, Casablanca, Morocco.* In this study, we report the experience of the nephrology-hemodialysis unit in UHC Ibn Rochd in Casablanca about the infant hemodialysis through 40 cases taken in charge from 1994 to the end of 1996. The mean age of the children has been 10 years old, with age brackets from 3 to 16 years old. The sex-ratio was 2/3. The predominant cause of nephropathy has been the classical glomerulonephritis (70%) then the chronical interstitial nephropathy (25%). The arteriovenous fistula constitutes the totality of the permanent vascular approach. No one was under peritoneal dialysis ambulatory (CPDA). The mean statural delay in 82% of the children is −2 DS. The cardiovascular complications dominated of the high blood pressure (40%), the pericarditis (17.5%), the cardiac failure (16%). 62.5% are protected against the viral hepatitis B (VHB), 10% suffer from viral hepatitis C positif (VHC+). The kidney transplantation has been achieved in three children. The rate of mortality in the hemodialyzed patients has been 20%.

Pregnancy in chronic hemodialysis outcome of multicentric women study. *H. Bahloul, K. Kammoun, K. Charfeddine, M. Ben Hmida, J. Hachicha, Department of nephrology. Hédi Chaker Hospital Sfax.—Tunisia.* Pregnancy is an infrequent event in hemodialysis women. Spontaneous abortion is frequent. It is exceptionnel to have viable baby. Our objectifs are to precise the frequency of pregnancy in women on hemodialysis and the complications in both mother and foetus. A preliminary questionnaire was sent to fifteen among nineteen dialysis units in south of Tunisia. The questionnaire asked for the number in all patients treated in the unit. The number of women of child bearing age (18 to 44 years) and those used contraception methods. When a pregnancy was reported, a more detailed questionnaire was sent to determine the senority of dialysis, residuel fonction (kidney); blood pressure control and the final outcome of these pregnancies. All units responded to the first questionnaire (100%). These units cared for 695 dialysis patients. 297 was the number of all women and 94 presented those of child bearing age (18 to 44 years), and only 14% of those were used contraception methods. Twelve (12) pregnancies were reported in eleven women (11); occurred during the survey period (Jan 1990 to Dec 1996). The frequency was 15%. The incidence of pregnancy in

our study was 5%. The foetus complications were observed in 83%. Successful ended pregnancy necessite to have some precautions: intensified hemodialysis, controlled the blood pressure, the correction of anemia, to have hemoglobin >7.5 g/dl by transfusion or erythropoietin. Since, mother and foetus's complication are frequent and dangerous, contraception must be used for dialysed women at child bearing age. The best contraception method for these patients is microprogestative pill.

Cystine: A risk factor in the precipitation of calcium oxalate calculi. *Martins M.C., Meyers A.M., Whalley N.A., Dept. of Medicine, Nephrology, University of the Witwatersrand and Metabolic Stone Clinic, Johannesburg, South Africa.* We noted that a number of patients who produced calcium oxalate (CaOx) calculi also had cystine (Cy) in their stones. 50% of these also had urinary Cy excretions resembling those found in heterozygous carriers but 50% did not. We also documented that urinary Cy levels (mg/24 hr) in normal black controls (n = 24) were significantly lower (44.4 ± 21.4) than in whites (n = 24) (71.2 ± 37.6); $p < 0.03$. Considering that less than 0.5% of the black population form recurrent calculi as opposed to 7% of whites, the question of the role of Cy in CaOx precipitation. 24 hr urine samples were collected in 9 normal controls and stored at 4°C. 3 specimens were collected at a time, pooled, spun and filtered (SFU) and ultrafiltered (UFC). Metastable limit (MSL) of urine was determined. Inhibitory activity was measured in UFU and SFU using 3 different concentrations of Cy. A Coulter Multisizer II was used to measure particle number, diameter and volume. Results were confirmed via the incorporation of ^{14}C-oxalate into the crystals. Scanning Electron Microscopy was used to further demonstrate the agglomerates. Cy caused both growth and aggregation of CaOx crystals, in a dose dependent manner. MSL of urine did not change with increasing doses of Cy, indicating that epitaxy is unlikely to be the mechanism involved in Cy induced CaOx precipitation. In conclusion, Cy results in marked enhancement of CaOx precipitation and this plus the epidemiological evidence of low urinary Cy levels in a non-stone forming population (SA blacks) together with the finding of Cy in CaOx stones in the non-cystinuric population, point to the possibility that urinary Cy may be a major risk factor in the formation of CaOx calculi.

Arterial hypotension in chronic hemodialysed patients. *Kheder A., El Matri A., Menjour A., Ben Hamida F., Abderrahim E., Ben Abdallah T., Ben Moussa F., Ben Maiz H., Department of Nephrology and Internal Medicine. (Pr H. Ben Maiz) Charles Nicolle Hospital, Tunis-Tunisia.* Arterial hypotension can occurs in chronic hemodialysed patients (CHD). In order to study (i) the frequency of hypotension in patients on maintenance hemodialysis and (ii) the role of sympathetic nervous activity in blood pressure regulation in uremic patients, we studied in 34 CHD (17 normotensives—NT—and 17 hypotensives—HOT), the duration of dialysis treatment, response to the valsalva maneuver (VM), plasma noradrenaline levels (NA), plasma renine activity (PRA) and plasma aldosterone concentration (PAC). NT and HOT were matched with regard to age and duration of dialysis (mean age 39.8 ± 2.3 year mean duration of dialysis 78 ± 6 months). Frequency of hypotension was 3.6%. By another way frequency of hypertension in the whole populations of CHD of Charles Nicolle's Hospital (n = 133) decreases when duration of hemodialysis is prolonged (73.8% of the patients were hypertensives when duration of hemodialysis was below 5 years VS 15.3% when duration of hemodialysis was above 10 years). During VM heart rate falls in 35% of HOT and in 58% of NT. NA levels were nearly similar in HOT and NT (224.29 ± 24.10 pg/ml VS 211.85 ± 20.21 pg/ml). Al through PRA was higher in CHD than in normal population, it was identical in the two groups of patients (HDT = 7.02 ± 1.28 pg/ml/h, NT: 6.95 ± 1.57 pg/ml/h, normal population: 0.2 to 0.28 pg/ml/h. Similar data were observed with PAC (HOT = 817.5 ± 105.2 pg/ml. NT = 630.3 ± 123.8 pg/ml. normal population: 10 to 150 pg/ml) We conclude that with increasing duration of dialysis treatment (i) baro receptors dysfunction occurs (ii) there is a resistance to the effect of vaso-constrictive hormones. This might be one important cause of arterial hypotension in patients on long hemodialysis treatment.

Lithogenic factors in black controls and black and white recurrent stone formers. *N.A. Whalley, M. Martins, M.I. Sonnekus and A. M. Meyers, Metabolic Stone Clinic, University of the Witwatersrand.* Renal stones are rare in the South African black population < 1%. Previously, the cause and composition of urinary stones in black and white South Africans differed, but are becoming increasingly similar. The aim of this study was to compare lithogenic risk factors in normal black men (BC, n = 12) and male black (BSF, n = 18) and white (WSF, n = 101) recurrent stone formers. Patients with cystinuria, MSK and hyperparathyroidism were excluded. Standard metabolic workup, comprising 2 × 24 hr urines, fasting serum and urinary ca:creatinine, was followed. There were no differences in serum Ca, Ca^{++}, PTH, and phosphate and urinary Na and Ox between the groups. Serum urate urinary(u) Ca, creatinine, phosphate and urate gave values WSF > BSF > BC. Urinary cirtrate was lowest in BSF < BC < WSF. Urinary volumes were higher in the SFs. The various categories of risk factors as found in the two groups (B:W) of SFs were as follows: Nil metabolic change 11%:9%, increased uCa due to increased uNa 11%:18%, renal hypercalciuria 6%:10%, absorptive hypercalciuria 6%:7%, dietary hyperoxaluria 17%:19%, mild metabolic hyperoxaluria 11%:21%, hypocitraturia 72%:58% and low volume in 17%:14%. Hypocitraturia only was found in 44% of BSF and 25% of WSF. Calculi available for analysis: BSFs had only CaOx whereas the WSFs had 82% CaOx, 16% CaPhos, and 2% uric acid. Although certain risk factors were similar, hyperoxaluria, dietary hypercalciuria, and renal hypercalciuria were more frequent in whites. Urinary hypocitraturia is the single most common cause in both groups. As urbanisation occurs, the urine of BSFs is becoming more like that of WSFs. Predictably nephrolithiasis will become more common in the black population.

Predictive factors of chronic renal failure in systemic lupus erythematosus. *Kammoun K., Hentati A., Bahloul H., Ben Hmida M., Charfeddine K., Bahloul Z., Jlidi R., Jarraya A., Hachicha J., Department of Néphrology. CHU—Hédi Chaker—3029 Sfax.* Renal involvement in lupus is frequent. It remains the major cause of morbidity and mortality in lupus erythmatous, despite immunosuppressive therapy. For precising the predictive criteria of outcome to chronic renal failure we reviewed all our cases of lupus. During the period 1985–1996 we observed 107 cases of lupus. All patients fulfilled four of the 11 American Rheumatism Association (ARA). 51 patients developed a lupus nephritis confirmed by renal biopsy. They were divided into two groups GI: patients with evolution to chronic renal failure GII: patients who until this report have a normal renal function. The were no difference among the two groups with regard to age, Sex, renal (edemia, proteinuria, hematuria) and extra renal symptoms. But hypertention and initial renal failure (Blood creatinine ≥ 150 μmol/l) cryoglobulinemia and diffuse proliferative lupus nephritis were more observed in group I. Treatment including pulse cyclophosphamide and Corticosteroids were more effective than steroids alone.

Intensive pulse therapy for focal segmental glomerulosclerosis in South African children. *Adhikari M., Bhimma R., and Coovadia H.M., Department of Paediatrics and Child Health, University of Natal, Durban, South Africa.* Focal segmental glomerulosclerosis (FSGS) is the most common form of steroid-resistant nephrotic syndrome in children and is increasing in incidence throughout the world. It is the second commonest cause of progression to end stage renal disease and is one of the most difficult management problems in both paediatric and adult nephrology units. Seven children with steroid resistant (SR-FSGS) were placed on a therapeutic protocol of methyl prednisone (MP), oral prednisone (MP), oral prednisone given over 18 months and oral cyclophosphamide (CYC) given for eight to twelve weeks (Regimen A). Another five children with SR FSGS were treated with a shorter course of MP (3 consecutive daily doses) intravenous CYC (monthly doses over 6 months) and oral prednisone 2 mg/kg (alternate days) (Regimen B). In Regimen A, one child had a short remission, in the remaining six, oedema subsided, the urine protein/creatinine ratio decreased, haematuria disappeared and the glomerular filtration rate (GFR) increased. The observation period was 21–42 months and the drugs were well tolerated. In Regimen B, two patients went into complete remission, one had partial remission. In these children, oedema cleared; the urine protein/creatinine ratio decreased; haematuria disappeared and the GFR rose. The follow-up was between 3–34 months. Minor

side effects were alopecia and transient hypertension. Of concern were severe infections in 2 children; one of whom died. Compared to Regimen A, Regimen B is six times cheaper with quarter the number of hospital visits. These observations may be of value in designing appropriate multicentre controlled trials, for the rational and optimum management of SR-FSGS.

Nephrotic syndrome in South African children revisited: Changing perspectives over 20 years (1976–1995). *Bhimma R., Coovadia H.M., and Adhikari M., Department of Paediatrics and Child Health, University of Natal, Durban, South Africa.* More than a quarter century ago, many centres in Africa had begun to identify unusual features of the Nephrotic Syndrome (NS) on this continent. We review our 20 year experience of 636 children with 23 (4%) were Coloureds (mixed race), 91 (14%) could not be categorised and were excluded from the analysis. Minimal change nephrotic syndrome (MCNS) continues to show the typical pattern among Indians: however it remains uncommon in Blacks. 134 of 168 (80%) Indians had biopsy-proven MCNS and 94% of these were steroid sensitive (SS); 60 of 65 (92%) had SSNS but without renal biopsy. Only 14% of Blacks had either biopsy-proven typical MCNS or SSNS without biopsy: a further 32 of 307 (10.4%) had MCNS lesions on biopsy but were steroid resistant (SR). The prevalence of focal segmental glomerulosclerosis (FSGS) appears to be increasing. Among Indians 443% had FSGS in 1995 compared to 2% in 1976. 28% of Blacks and 44% of Coloureds in 1995 had FSGS compared to 5% Blacks in 1970 (we had no Coloureds then). Only 4% of these patients were SS; none of whom were Blacks. Membranous nephropathy is the commonest type of lesion seen in Blacks accounting for 40%; 86% were associated with hepatitis B antigens. 7 of 286 (2.4%) Indians and 6 of 23 (26%) Coloureds had idiopathic membranous nephropathy; 15% were SS. Proliferative lesions were uncommon in all population groups, present in 6% of Blacks; 5% Indians and 9% Coloureds in 1995 compared to 33% Blacks and 15% Indians in 1970. Overall mortality was 3%. In brief, this is the largest reported series of NS among children in Africa and shows a typical pattern in Indians, and unusual frequency of histological types in Blacks and an intermediate picture in Coloureds.

Insulin resistance in a group of hypertensive Cameroonians. *Youmbissi T.J., Mbanya J.C., Tokpanou E., Departments of Nephrology and Endocrinology Medical School, Yaounde, Cameroon.* Insulin resistance and Hyperinsulinaemia were studied in 17 non obese newly diagnoses Cameroonian Hypertensives with normal glucose tolerance and an equal number of matched normal glucose tolerant normotensives. Both groups showed normal and comparable a) mean fasting serum insulin levels (5.9 versus 4.6 micro IU/ml) b) mean fasting glucose/insulin ratios (0.67 versus 0.78) c) mean basal serum C peptide levels (0.67 versus 0.78 ng/ml) d) mean stimulated serum C peptide levels (4.40 versus 4.05 ng/dl) e) mean fasting Insulin/C peptide ratios (3.4 versus 5.6) Both groups showed no significant variation in the insulin areas under curve (144 versus 146) after ingestion of glucose, the mean Glucose/Insulin ratio followed almost the same curve in both groups. No correlation was found in hypertensives between a) Fasting blood glucose and systolic blood pressure (SBP) ($R = 0.28$), diastolic blood pressure (DBP) ($R = 0.29$) or mean blood pressure (MBP) ($R = 0.31$) b) Fasting serum insulin and SBP ($R = 0.29$). DBP ($R = 0.31$) c) Fasting Glucose/Insulin and SBP ($R = 0.20$) DBP ($R = 0.14$) or MBP ($R = 0.22$). Hypertensives, had higher serum cholesterol ($P = 0.01$) lower HDL cholesterol ($P < 0.01$) but similar serum Triglycerides and apolipoprotein A-1 as normotensives. Our study did not confirm Ferrannini's hypothesis that essential Hypertension could be an insulin resistant state independent of obesity or non insulin dependent diabetes mellitus. It showed however the usual association between Hypertension and altered serum lipid levels.

Hypertension in Tunisian black population. *M. Ben Hmida, G. Brillet*, M. Chtara, J. Hachicha, K. Charfeddine, A. Jarraya, Dpt. of Nephrology, H Chaker Hosp. Sfax Tunisia & *Necker Hosp., Paris, France.* According to US data, the prevalence of hypertension (H) is greater in black (B) adults than in white. The reasons for the apparent increased susceptibility of American B to develop H and its target organ complications are not well understood. It has been proposed that, in B, lower educational level and reduced access to medical care my contribute to the development of these target organ complications. In Tunisia, when socio-cultural and economic factors contributing to H do exist, no correlation with ethnical origin is found. We have prospectively investigated two groups of hypertensive adult patients, G1: 100 B pts (50 M + 50 W) and G2: 100 white pts (50 M + 50 W). This two groups were matched regarding social and geographical origin, educational level, occupation, physical activity, matrimonial and genital activity for women, tobacco and alcohol habits, medication and family history of diabetes mellitus and H. Our results read as follow:

	White	Black	*P*
Age (*yr*)	51 ± 11	50 ± 12	NS
Obesity (*BMI* > *25*)	65%	78%	<0.05
Anti-HT treatment			
Monotherapy	61%	47%	<0.05
Bitherapy	38%	41%	NS
Tritherapy	1%	12%	<0.01
Complications			
Coronary disease	2%	11%	<0.001
Left VH	4%	11%	<0.02
Pl Cr (*mmol/l*)	79 ± 30	115 ± 68	<0.03
Cr Cl (*ml/mn*)	110 ± 40	88 ± 42	<0.001
U Pr (*g/24 h*)	0.17 ± 0.1	0.53 ± 0.15	<0.001
Stroke	1%	7%	<0.001
Retinopathy II/III	0/2%	4/28%	<0.001
Blood pressure control			
Well	49%	9%	<0.001
Mild	49%	65%	<0.001
Uncontrolled	2%	26%	<0.001

Our study shows that in the absence of difference in socio-cultural and economic factors, H in B seems to be more severe with frequent target organ complications than in white. The role of genetic rather than socio-economic factors is likely to be implicated.

End-stage renal disease of the Tunisian child: Epidemiology and etiologies. *A. Kamoun*, F. Jawahdom*, T. Ben Abdallah**, F. Ben Moussa**, J. Hachicha***, H. Ben Maiz**, R. Lakhoua*, Department of Pediatrics, Charles Nicolle's Hospital, Tunis. Department of Nephrology, Charles Nicolle's Hospital, Tunis. Department of Nephrology, Hedi Chaker's Hospital, Tunis, Tunisia.* Data on the epidemiology and etiologies of pediatric renal failure in northern Africa are scarce and basically concern subjects who have not reached end-stage renal disease (ESRD). The aim of the present study was to establish the incidence, the characteristics and the etiologies of ESRD among Tunisian patients under 15 years. **Patients and methods:** Our study includes 134 Tunisian children with ESRD admitted for taking in change by dialysis. Sixteen children were less than 5 years and 43 were aged between 5 and 10 years. The sex ratio (M/F) of the children was 1.91. **Results:** The estimated incidence of pediatric ESRD in Tunisia is 7 new cases per year and per million child population under 15 years. The chief etiologies of ESRD are glomerulonephritis (19%), hereditary nephropathies (29%), malformative uropathies (16.5%), renal hypoplasia (4.5%) and amyloidosis (4.5%). The etiology of ESRD was undetermined in 26% of cases. Primary hyperoxaluria (18 cases) represent the most important etiologal group among hereditary nephropathies. **Conclusion:** Our findings, compared with the European data, show a particularly high frequency of primary hyperoxaluria (13%) and an unusual proportion of male subjects.

The problem of end stage renal failure in the indigenous populations of Australasia. *D.J. Pugsley, A.E. Seymour, W. Hoy, Renal Unit, The Queen Elizabeth Hospital, Woodville, South Australia 5011.* Australian Aborigines (AA) and the Maori and Pacific Islander (MPI) population of New Zealand (NZ) are known to harbour rates of serious renal disease that are much higher than those found in other residents of Australia and NZ. Between the ages of 20 and 50, the entrance of AA

onto end stage renal failure (ESRF) programmes is at least 10 times that of other Australians. MPI constitute half of all patients entering ESRF programmes in NZ, although they only comprise 13% of the population. The prevalence of non-insulin dependent diabetes mellitus (NIDDM) is high in these populations and diabetic nephropathy is a major contributor to the burden of ESRF. The control of hypertension and hyperglycaemia is difficult to achieve, and the rate of progression of renal failure is often rapid. Among the causes of glomerulopathy (G) in AA there is a striking incidence of glomerulomegaly which progresses to ESRF by way of increased glomerulosclerosis. Mesangiopathic G is differently distributed and mesangio-capillary GN is over represented when compared with the non-Aboriginal population. Membranous G has rarely been reported. Preliminary research suggests that: renal disease later in life may be associated with obesity developing against a background both of low birth weight and repeated infections. How this may be linked to the observed pattern of pathology is not yet known. The treatment of ESRF by dialysis and transplantation is difficult for cultural, social, geographic and economic reasons, and the survival of AA is significantly worse than that found amongst other Australians with ESRF.

Acute renal failure for children (74 cases). *Fatihi E., Hachim K., Benghanem Gharbi M., Zahiri K., Ramdani B., Zaïd D., Service de néphrologie-hémodialyse, CHU Ibn Rochd, Casablanca, Morocco.* A retrospective study of 74 files of acute renal failure (ARF) of the child collected from 1990 to 1996 in the department of Nephrology-Hemodialysis of U.H.C. Ibn Rochd in Casablanca permitted to analyse the etiologic and prognostic factors of this affection. If the positive diagnosis doesn't make problems, the determination of the causes and the physiopathologic mechanism is difficult. The etiologies of the ARF of the child were dominated by the glomerular nephropathies (60.8%) which the acute post-infections glomerulonephritis represent the majority (40.5%) followed by hemolytic and uremic syndromes. The factors of poor prognosis were represented by etiologic circumstances (Obstruction), the etiopathogenic factors (the infection), the young age of the child (newborn and baby), the oligoanuria, the existence of complications with septicemic type, the digestive hemorrhage, the severe neurologic disturbances, the pneumopathies, the important retention of salt and water into the body overload or the metabolic acidosis. The precocious and frequent hemodialysis associated to symptomatic and etiologic treatment permitted to have a rate of good results equal to 89.1%, this rate in comparison with data of literature is satisfactory. The real amelioration of the prognosis can be obtained only if a preventive action aiming at the treatment of etiopathogenic factors which can cause the acute renal failure and a best knowledge of this pathology by the nephrologist.

Significance of microalbuminuria in Type II diabetic patients. *F. El Younsi, K. Khiari, N. Ben Abdallah, T. Ben Abdallah, H. Ben Maiz, Department of Nephrology and Internal Medicine (Pr H. Ben Maiz), Charles Nicolle Hospital—Tunis—Tunisia.* In insulin dependent diabetes (IDDM), microalbuminuria (malb) predicts renal and cardiovascular disease. We report the result of malb determination in 50 type II diabetic patients (21 men, 29 women) compared to 30 non diabetic controls (NDC). Urinary albumin levels were measured by radioimmuno-turbidimetry. Mean malb concentration was less than 5 mg/l in NDC versus 18.2 mgr/l in NIDDM. There was a significant increase of urinary albumin excretion (UAE) rates in NIDDM with high blood pressure. Correlation was also positive between malb and body mass index (BMI) and lipid abnormalities, essentially triglycerids. Correlation between malb and duration of diabetes was negative. These results showed a positive correlation between UAE and several cardiovascular risk factors like obesity, hypertension and hyperlipidemia, malb could be considered as a predictor of macrovascular complications in NIDDM patients. Hyperinsulinemia is frequently present in this situation known as syndrome X.

Type 1 primary hyperoxaluria: Tunisian experience about 31 pediatric cases. *Kamoun A.*, Abdelmoula J.**, Daudon M.***, Jawahdou F.*, Zghal A.**, Ben Ammar S.**, Belkahia C.**, Lakhoua R.*, *Service de Pédiatrie, Hôpital Charles Nicolle, Tunis; **Laboratoire de Biochimie, Hôpital Charles Nicolle, Tunis; ***Laboratoire de Biochimie et INSERM U 90, Hôpital Necker, Paris.* We report on 31 children (16 boys) presenting with type 1 primary hyperoxaluria (PH1). The mean age at diagnosis was 6.1 years (range: 3 months–14.8 years) and the average follow-up period was 25 months. The mean delay between first symptom and diagnosis was 1.3 year. The diagnosis of PH1 was carried out by determination of oxaluix in 16 cases, by renal histology in 11 cases and by physical analysis of urolithiasis in 9 cases. In the 9 cases where urinary glycolate was assayed, the level was found high. At the time of diagnosis the renal function was normal in 10 children, moderately altered in 1 and severely so in 20. During the follow-up the renal function remained stable in 10, greatly improved in 2, deteriorated in 5. The 14 patients who experiences end-stage renal disease at the diagnosis remained unchanged. Urinary stones were present in 23 patients among 24 aged more than 2 years and in one among 7 infants. A response to pyridoxin therapy was noticed in 2 patients. Extracorporeal shock wave lithotripsy performed in 7 patients was efficient in only 3. In 9 patients oxalate bone disease was correlated with both renal function and dialysis duration, whereas retinal involvement observed in 7 patients was not.

Karyomegaly of tubular cells as early stage marker of the nephrotoxicity induced by Ochratoxin A in rats. *Achour A.*, Maaroufi K.**, Zakhama A.*, Bouraoui S.*, Elmay M.*, Bacha H.**, *Service de Néphrologie CHU Monastir—TUNISIE; **Laboratoire Toxicologie—F.M. de Monastir—TUNISIE.* Cases of Karyomegaly were described by Sclare and by Mihatch in patients affected with tubular-interstitial nephropathy. The Karyomegalic cells showed enlarged nuclei with accumulation of genetic material. No aetiology was suggested. Our study in rats experimentally intocicated by ochratoxin A, a well-known nephrotoxic compound, indicates the presence of Karyomegaly with alteration of the tubular tissue. In control animals no karyomegalic cell was detected. These observations suggest that karyomegaly with megacytosis may be caused by the nephrotoxic ochratoxin A in the kidney. In addition abnormal mitosis together with karyomegalic cells were observed at ealier stage of the intoxication (30 days) suggesting possible regeneration in the OTA insults are stopped. After 90 days of treatment, the degeneration increased and only karyomegalic and apoptotic cells were observed indicating that the regeneration does no more occur and that the regeneration become irreversible.

Congenital/infantile nephrotic syndrome with pulmonary stenosis—A new syndrome? *Kala U.K., Jacobs D.W.C., Verhaart S. and Goetsch S., Department of Paediatrics, Chris Hani Baragwanath Hospital, University of the Witwatersrand.* **Aim**: Analyse patients presenting with congenital/infantile nephrotic syndrome (CNS) with associated pulmonary stenosis (PS) with regards to clinical features, laboratory and histological data and natural history. **Methods**: Descriptive study. **Results**: Ten patients with a mean age of presentation 11 $\pm$ 9.8 months, 5 males and females with sets of siblings, and one with a family history of affected siblings. All patients having a degree of hyperteliorism, profuse sweating, flattened nasal bridge, soft ear pinnae, hepatosplenomegaly, bilateral nephromegaly and gross nephrosis. The mean gradient across the pulmonary valve echocardiographically being 35 $\pm$ 14.3 mm Hg, the higher gradient having right ventricular heave—but all having pulmonary ejection systolic murmur. Histologically varying degree of focal glomerulosclerosis with segmental and or global fibrosis with cyst formation in all except one. Associated varying intensity of interstitial fibrosis, the arterioles appearing hyperplastic. On electron microscopy (EM) all have thin membranes for age and Alport like changes of lamination and rarefaction with loss of lamina densa. Two out of ten went into renal failure after a mean of 22 months. Four demised two from gastroenteritis, two from renal failure and one of which had cerebral thrombosis and the other chickenpox. Two lost to follow up. Presently four patients alive with a follow up 21.3 $\pm$ 17.8 months—all still nephrotic. **Conclusion**: All patients presented having distinct clinical features and pathology. A variant of Alport's or a defect in collagen or other basement membrane protein not described yet?

Intravenous iron sucrose on haemodialysis—A multicentre clinical study. *R. Van Zyl-Smit, M.R. Moosa, C.D. Potgieter, H.G. Viljoen, S. Naiker, Renal Unit, Groote Schuur Hospital Dept. Medicine, University of Cape Town.* Five South African haemo-dialysis units participated in a study designed to evaluate the tolerance, safety and efficacy of an intravenous iron sucrose preparation (Venofer®). A total of 131 patients were included in the safety, and 105 in the efficacy analysis. All patients had a haemoglobin level of ≤10g/dl, a serum transferrin saturation of ≤20% and serum ferritin of ≤200 ng/ml. The drug was administered during dialysis at a dose of 5 ml (100 mg), three times per week till a pre-calculated cumulative dose was reached. It was extremely well tolerated with the most frequent side effect possibly related to the drug being "hypotension" reported in 13% of cases. The serum ferritin rose from a mean baseline of 74 ng/ml to 486 ng/ml by week two, TIBC from 54 μmol/l to 44 μmol/l and % transferrin saturation from 13.6% to 25.8%. The mean baseline haemoglobin was 7.3 g/dl SD 1.6, it rose to 9.2 g/dl SD 1.8 by week two and 9.2 g/dl SD 1.9 during the observation period, the haematocrit from 22.5% to 28.3% and 28.6%, the MCV from 83.1 fl to 89.6 fl and 89.9 fl, the MCHC from 32.1 g/dl to 32.6 g/dl and 32.5 g/dl. **Conclusions:** Venofer® appeared to be perfectly safe with none of the possible clinical side effects being different from those expected during uncomplicated haemodialysis. No adverse biochemical reactions were observed. The rise in haemoglobin levels, haematocrit and MCV was highly significant, whilst all parameters of iron status were changed in a positive way.

Renal biopsies of HIV positive patients at Baragwanath Hospital 1989–97. *L. Pantanowitz, S. Goetach, O. Butler, I.J. Katz, Baragwanath Hospital, Dept. of Nephrology.* HIV associated nephropathy (HIVAN), more common in black patients, simultaneously affects glomeruli, tubules and interstium. The nephropathy is characterized histologically by focal segmental sclerosis and hyalinosis (FSH) and tubulointerstitial nephritis (TIN). The aim of this study was to retrospectively characterize HIVAN seen at Baragwanath hospital and determine a possible immune-mediated aetiology. Renal biopsies of 21 black HIV positive patients (mean age 29 years, 71% female) from 1989 to 1997 were reviewed. Histopathological findings in all cases revealed concomitant glomerular and tubulointerstitial pathology. Glomerular disease was predominated by FSH (62%). Mesangial proliferative, membranous, and post infectious GN each accounted for 14% of histopathology. Membrano-proliferative GN was found in 5%. Eighty one percent of biopsies showed TIN. Acute tubular necrosis was present in 48% of biopsies and pyelonephritis in 14%. Renal tuberculosis was found in one case. Immunflouesence revealed IgM (31%) and IgG (19%) positive in capillary tufts, deposition of IgA (38%) and albumin (25%) in tubular casts and cells, and C3 (44%) in the mesangium and sclerotic areas. Glomerular deposits were seen with regularity using TEM. Endothelial tubuloreticular inclusions were found in two cases. These results are consistent with previous reports of a 'pan kidney' pathological involvement in HIVAN. They confirm the frequency of FSH and TIN in these patients. Infectious complications are uncommon. Histopathological findings suggestive of an immune-mediated aetiology warrant further study in South Africa.

Efficacy and safety of Cilazapril (CLZ) versus hydrochlorothiazide (HCTZ) and a combination of Cilazapril/Hydrochlorothiazide (CLZ/HCTZ) in a group of black Africans with mild to moderate hypertension. *Youmbissi T.J., Department of Nephrology Medical School, Yaounde, Cameroon.* The aim of this study was to find out whether CLZ alone or in combination was an efficacious and safe treatment in black African hypertensives. This was a double blind, multicenter randomised three arm parallel group study with a 2 week single blind placebo run-in period to the 8 week double blind treatment. Test drug dosage was 2.5 mg for CLZ, 12.5 mg for HCTZ and CLZ 2.5 mg/HCTZ 12.5 mg for the combination. Patients previously treated with symptomatic drugs underwent a 4 week wash-out period. After the screening at week −2, patients began a 2 week placebo run-in and selection period. Patients with a sitting DBP of >95 mm Hg, and <155 mm Hg at week 0 entered the active treatment period. If the sitting DBP was >90 mm Hg, at 4 week, the medication was doubled. Clinical and laboratory evaluations were performed at weeks –2, –1, 0, 4 and 8. χ^2 test and analysis of variance were used. 106 patients were recruited (54 women and 52 men, mean age: 44.1 years, SD: 10.26); 8 patients were excluded. Of the remaining, 33 were on CLZ, 32 on CLZ-HCTZ and 33 on HCTZ. The dosage was doubled from week 4 in 17 patients on CLZ (51.5%) 9 patients on CLZ-HCTZ (28.1%) and in 18 patients (54.6%) on HCTZ. Response rates (DBP < 90 or a decrease of DBP from baseline > 10 mm Hg, at week 8 were the following: 24 patients (72.7%) on CLZ, 21 (65.6%) on CLZ-HCTZ and 23 (69.7%) on HCTZ. There was no significant difference. But at week 4 responses were: 16 patients (48.5%) on CLZ, 23 patients (71.9%) on CLZ-HCTZ and 17 (51.5%) on HCTZ. This improvement of the response rates from week 4 to week 8 in the CLZ-HCTZ groups is due to the fact that the dosage had to be doubled in 51.5% in the CLZ and 54.5% in the CLZ and 54.6% in the HCTZ groups whereas only 28.1% in the CLZ/HCTZ group needed the double dose. 9 adverse events CLZ-HCTZ group and 2 in 2 patients (5.7%) in the HCTZ were observed. Coughing was the most frequent event (4 patients on CLZ, 1 on CLZ/HCTZ, and 0 on HCTZ). No serious adverse event could be observed. There was no significant change in the clinical or laboratory variables during the study. Cilazapril alone or better in combination with hydrochlorothiazide is an efficacious and safe treatment in Black African Hypertensives.

Detection of *Oxalobacter* in the human gastrointestinal tract. *Arvinda Sooka,[1] Anthony M. Smith, Anthony M. Meyers[2] and Hendrik J. Koornhof, [1]South African Institute for Medical Research, Johannesburg, [2]Dept. of Nephrology, Johannesburg Hospital.* Anaerobic *Oxalobacter* species have been known to metabolise oxalates in the colon of normal people. We postulate that a deficiency of these bacteria may be responsible for the production of oxalate kidney stones and an abundance of these bacteria may protect Black South Africans from oxalate calculi. The aim of the study was to detect *Oxalobacter* organisms in stools of black males. The anaerobic technique used to culture the bacteria was similar to that described by Hungate using a roll tube method selecting for oxalate-metabolizing bacteria. Since *Oxalobacter* is difficult to culture, we designed a PCR technique to detect *Oxalobacter* OXIT gene from stool specimens. The *Oxalobacter* was cultured in an anaerobic broth for broth for 48 hours. The cells were harvested and used in a 50 μl PCR reaction to amplify a ≈795 bp DNA fragment using appropriate PCR primers. To determine the specificity of the PCR, we analyzed DNA from eleven bacterial species commonly present in faeces. No products were amplified in these control organisms giving a 100% primer specificity. A commercial kit was used to extract DNA from stool specimen which were seeded with *Oxalobacter*. PCR successfully demonstrated *Oxalobacter* in these spiked specimens. By estimating the PCR product, a semi-quantitative method will be devised to measure the quantity of *Oxalobacter* in various population groups and stone formers.

Mitochondrial cytopathy in Fanconi syndrome. *R.D. Gilbert, M. Emms, C. Sinclair-Smith, M. Shuttleworth, Red Cross War Memorial Children's Hospital, Private Bag Rondebosch 7701.* Mitochondrial abnormalities are now recognised as a cause of Fanconi syndrome. We describe 2 children, 1 with Pearson's syndrome, with Fanconi syndrome and bizarre mitochondria in the renal tubular cells. A 21-month-old boy was referred because of fever and dehydration. He was underweight, pale and had a 2 cm firm hepatomegaly. Both kidneys were massively enlarged. There was laboratory evidence of generalised proximal tubule dysfunction, elevation of blood lactate and pyruvate and mild macrocytic anaemia. The bone marrow showed dyserythropoeisis with vacuolated granulocyte precursors. Renal and muscle biopsies showed highly abnormal mitochondria. He died shortly afterwards of uncontrollable metabolic acidosis. Necropsy confirmed pancreatic atrophy. A 10-year-old girl was referred because of poor response to treatment of Fanconi syndrome diagnosed at 7 years; no cause had been found. She had severe rickets, muscle wasting and weakness. Investigations confirmed Fanconi syndrome with elevated lactate and pyruvate. Renal and muscle biopsies showed abnormal mitochondria. She has improved on more aggressive management. Mitochondrial abnormalities need to be considered in the differential diagnosis of Fanconi syndrome. Renal biopsy with electron microscopy may be indicated in some patients.

Acute renal failure in children in the Congo. *Assambo-Kieli C., Assounga A.G., Mafoua A., Nzingoula S., Brazzaville's University Hospital and Marien Ngouabi University, Brazzaville, Congo.* Acute renal failure (ARF) is a common pathology in the Congo. This is a 6 year retrospective study aiming at analyzing the epidemiology and treatment modalities of ARF in Brazzaville's University Hospital from 1989 thru 1994. One hundred and five cases of ARF including 54 boys (51.4%) and 51 girls (48.6%) have been recorded out of 10,512 children admitted in the Pediatrics Department (0.99%). ARF represents 13.09% of 802 kidney patients. The main etiologies of ARF include: acute gastroenteritis with dehydration (25.7%), nephrotic syndrome (17.4%), sepsis (15.23%), malaria (12.38%) and acute glomerulonephritis (9.5%). Most cases were treated with conservative measures while peritoneal dialysis (PD) was used in 8 cases (7.62%). The outcome of ARF was: recovery in 50.4% lethality in 37.14% and chronic renal failure was found in 9.52% of cases. The high lethality rate and the high cost of PD render urgent the need for preventive measures.

The clinico-pathological manifestations of renal disease in HIV positive patients, Tygerberg Hospital. *A.A. Walele, W.D. Bates and M.R. Moosa, University of Stellenbosch, Cape.* Three characteristic patterns of HIV related renal disease HIV-associated nephropathy (HIVAN), immune-complex glomerulonephritis (IGGN) and tubulo-interstitial nephritis (TIN) are reported. The aim of the study was to document the clinical manifestations of renal disease in HIV positive patients who underwent renal biopsy at Tygerberg Hospital. Since 1990 nine renal biopsies in eight HIV positive patients, mean age 29 years, have been undertaken. Only one patient had AIDS defining criteria. Four patients with histological features characteristic of HIVAN had nephrotic syndrome and renal failure (Cr $>$ 120 μmol/L) was present in three of them. Another three patients with ICGN presented with nephritic/nephrotic syndrome, low complements and renal failure. Biopsy showed TIN in one patient with renal failure. Two patients were dialysis dependent. Histology confirmed HIVAN in one and ICGN in the other who progressed to crescentic glomerulonephritis on follow-up renal biopsy. This small renal biopsy series in HIV positive patients documents three histopathological patterns. A typical clinical presentation was nephritic/nephrotic syndrome with renal failure.

Antimicrobial acute renal failure (ARF). *Ben Dhia N., Chaabouni M., Bouraoui S., Frih A., Mahjoub S., Elmay M., Nephrology Dep. CHU Monastir 5000—Tunisia.* The most serious clinical manifestation of antimicrobial nephrotoxicity is the occurence of ARF. It's difficult to link ARF to a particular antibiotic other causes usually exist and contribute to renal damage. Authors report a retrospective study of 26 patients where ARF was due to gentamicine 18 cases, penicilline 4 cases, oxacilline 1 case, colimycine 1 case, Rifampicine 1 case and vancomycin 1 case. Ages varied from 15–83 years (Mean 50.8), sex ratio: 1. Serum creatinine was in normal ranges before antimicrobial treatment in 92%, 75% of our patients were nonoliguric. ARF was mild to moderate in 19 cases (73%) and severe in 7, hemodialysis sessions were indicated in 2 patients and renal biopsies in 2 cases. Imputability of ARF to antimicrobial agents was established on clinical criterions: Frank increase of serum creatinine after initiation of the drug, coexistence of allergic signs, or risk factors of nephrotoxicity. We conclude that critically ill patients, who often have a marginal renal blood flow and/or additional factors predisposing to antibiotic nephrotoxicity must be monitered.

Nephrotoxicity of non steroidal anti-inflammatory drugs (NSAID). *Ben Dhia N., Chaabouni M., Frih A., Bouraoui S., Touzi M., Bergaoui N., Elmay M., Nephrology Department—CHU Monastir 5000—Tunisia.* There are several reports on nephrotoxicity of NSAID. They act mainly by reducing the production of prostaglandins and thromboxane, which are vasodilatators particularly important in renal hypoperfusion situations (hypovolemia, cardiac failure . . .). In 10 years (1986–1996) 17 patients presented to our unit in renal failure associated with the use of NSAID. Ages varied from 26–76 years (Mean 55.3). A skin rash was reported in 4 cases suggesting allergic basis. 14 patients (82.5%), were oliguric and 3 anuric. Predisposing factors such as history of chronic renal failure advanced age (70%), heart failure (11%). Aggressive diuretic therapy (11%), diabetes (11%), and association of more than 2 factors was noted in 70% of our patients. Mean durations of NSAID therapies were 32.5 days fordiclofenac and 47.9 days for indomethacine. Acute renal failure was severe in 5 patients (29%) and necessitate a temporary hemodialysis sessions in 2 patients (11.9%). Proteinuria was positive in 11 cases (61%) (Mean 2.18 g + 1.1/day). In 15 patients ARF greatly improve when NSAID was stopped, although 2 patients with CRF were placed in HD programm.

Allograft membranous nephropathy: Favourable response to high dose steroids. *A.A. Khad, P Johnston, D.M. Lewis, A.M. Davison, Department of Renal Medicine St James's University Hospital, Leeds, UK.* Recurrent membranous nephropathy in renal allografts is infrequent. Twelve cases were reported before 1982 and a further 21 between 1982 and 1995. De novo membranous nephropathy is more common, with an incidence of 1–2%. The principle manifestation is proteinuria and progressive loss of graft function. Intensification of immunosuppressive regimes has had little impact on graft survival. We report 4 patients, with nephrotic range proteinuria secondary to allograft membranous nephropathy, in whom remission followed high dose steroid therapy. The first patient was a 43-year-old man transplanted in 1990 (original disease small kidney: ? familial glomerulonephritis). Immunosuppression included Prednisolone, Axathinoprine and Cyclosporin and good graft function was achieved. Cyclosporin was stopped after 16 months. Three months later the patient developed proteinuria ($>$11 g/day). Serum creatinine remained normal. An allograft biopsy revealed membranous nephropathy. The patient was treated with 3 boluses of intravenous Methylprednisolone (0.5 g) and then oral Prednisolone, 125 mg alternate days for four months. Proteinuria dropped to 0.5 g/day, and serum creatinine remained normal. The second patient was a 51-year-old women patient transplanted in 1989 (original disease bilateral small kidneys). Normal graft function was achieved on Cyclosporin monotherapy. After 6 months she was converted to Prednisolone and Azathioprine. Twelve months later she developed proteinuria (12 g/day). Allograft biopsy revealed membranous glomerulonephritis. The patient was treated with 3 boluses of intravenous Methylprednisolone (1 g), and 125 mg Prednisolone alternate days for four months. Proteinuria dropped to $<$0.5 g/day and serum creatinine remained normal throughout. The third patient was a 52-year-old man transplanted in 1988 (original disease chronic glomerulonephritis). Immunosuppression included Prednisolone, Azathioprine and Cyclosporin and normal graft function was achieved. Cyclosporin was withdrawn after 24 months. Thirty months post-transplant, the patient developed 4.5 g/day of proteinuria. Serum creatinine remained normal. Allograft biopsy revealed membranous nephropathy. The patient was treated with 3 boluses of intravenous Methylprednisolone (1 g) and oral Prednisolone, 125 mg alternate days for two months. Proteinuria declined rapidly to 0.5 g/day. The fourth patient was a 29-year-old man transplanted in 1990 (original disease focal proliferative glomerulonephritis). Normal graft function was achieved on cyclosporin monotherapy. After 14 months he was converted to Prednisolone and Azathioprine. One month later the patient developed proteinuria (7 g/day). Allograft biopsy revealed membranous nephropathy. The patient was treated with 3 boluses of intravenous Methylprednisoline (1 g) and 125 mg Prednisolone alternate days for five months. Proteinuria declined to 1 g/day and serum creatinine remained normal throughout. Contrary to previous reports, we found high dose steroids to be effective in suppressing nephrotic range proteinuria in allograft membranous nephropathy.

Analysis and value of angiography in live related renal transplantation. *A.A. Haffejee, K.S. Satyapal, B. Singh, L. Ramsaroop and S. Naicker, Department of Surgery and Medicine, University of Natal.* **Introduction:** In diagnostic work-up of renal patients, it is necessary to be familiar with blood supply of kidneys and number of arteries supplying each kidney. Renal angiography remains gold standard in pre-operative investigation. Study aimed to evaluate accuracy of pre-operative angiography reporting compared to intra-operative findings. **Patients and Methods:** Retrospective analysis of 61 angiograms of live related donors performed at Addington Hospital was compared to intra-opera-

tive findings of donor and recipient between 1993–1996. Age Range: 22–53 years; Sex: 26 males, 35 females; Race: 33 Indians, 16 Africans, 7 Whites, 5 "Coloureds"; Relationship: siblings: 33; parent-child: 15; husband-wife: 9; relatives: 4. Angiography performed using Seldinger technique with femoral artery puncture. PA and lateral films taken. Reporting performed by radiologists with varied experience. Surgery performed by team of surgeons. Intra-operative findings correlated with radiological reports. **Results:** Additional arteries encountered in 15 patients [2: 12 patients; 3: 3 patients]. Six radiological reports did not correlate with operative findings (4 reported single arteries: 2 present; 2 reported dual arteries, 3 present). Anastomotic time (minutes): one artery: 19; two arteries: 26; three arteries: 36. Post-operative course of patients with multiple arteries was not associated with increased morbidity. **Conclusion:** Incidence of multiple renal arteries is relatively high. Clinical use of kidneys with up to three arteries is not associated with increased morbidity. Accuracy of reporting of angiography is 91%.

The kallikrein-kinin system in renal disease. *Naicker S., Ramsaroop R., Moodley D., Naidoo S., Bhoola K.D., Dept. of Med., Pathology and Pharmacology, University of Natal Med. School, Durban, S. Africa.* The renal kallikrein-kinin system is involved in inflammation, blood pressure regulation and sodium and water homeostasis. Tissue kallikrein (TK) status was studied in 16 patients with renal disease and 12 normal subjects: urinary TK activity was measured by an amidolytic method, TK and kinin B2 and B1 receptors localised in renal tissue. Urinary TK excretion was significantly decreased in all patients with renal disease and more markedly so in severe renal failure. Reduction of immunolabelled TK was noted in the distal nephron in renal disease. Receptor status in normal kidney showed B2 reactivity in the entire nephron, whereas no expression of B1 was seen. In renal disease there was an upregulation of B1 and a downregulation of B2 receptors. These observations indicate a probable role for the kallikrein kinin system in renal disease and may contribute to its manifestations such as hypertension.

	Urinary TK *ng/μg protein*		
		Renal disease	
	Control	Severe	Mild
n	12	8	8
x ± SEM	78.9 ± 31.7	1.8 ± 0.7	16.6 ± 6.7
P		<0.01	<0.05

Localisation of tissue kallikrein in the kidney of black African women with early onset pre-eclampsia—A pilot study. *T. Naicker, S.M. Khedun, J. Moodley, K.D. Bhoola, EM Unit, Department of Pharmacology and MRC Pregnancy Hypertension Research Unit, Natal Medical School, Durban.* Increased renal production of vasodilator mediators like kinin would counteract the vasospasm of pre-eclampsia. This study examines the cellular localisation of tissue kallikrein (TK), a potent kinin forming enzyme within the nephron of patients with early onset pre-eclampsia. Using the peroxidase-antiperoxidase immunoenzyme complex, TK was immunolocalised in the principal cells of the distal connecting tubules and cortical collecting duct cells of the distal nephron of the control tissue. Moderate reactivity was observed in the epithelial lining the Bowman's capsule. In early onset pre-eclampsia, TK was additionally localised in the proximal tubule cells. In patients with hypertension in pregnancy, the occurrence of TK in the proximal tubule suggests either gene induction or emiocytosis.

Profunda femoris artery to saphenous vein PTFE vascular access in patients on long term haemodialysis. *Mirza K., Pontin A., Kahn D., Department of Surgery, University of Cape Town Medical School, Observatory 7925, Cape Town, South Africa.* Arteriovenous fistula, introduced by Brescia and colleagues has become the primary form of vascular access for long term haemodiaysis. However in approximately 10% of patients this is either not possible or has already been exhausted and other forms of vascular access are required. Both biological and synthetic grafts are available. The expanded polytetrafluorethylene (e-PTFE) is currently the most popular alternative. Between 1993 and 1997, 10 patients underwent formation of thigh vascular access by insertion of PTFE graft between profunda femoris artery and saphenous vein. These were 4 males, and 6 females, mean age 39.7 yrs (range 27–55 yrs) and mean duration of renal failure 8.5 years (range 5–15 yrs). All patients had multiple previous vascular accesses and were assessed pre-operatively for evidence of peripheral vascular disease. Immediate success rate was 100% and all grafts were still patent after 6 months follow-up (range 1–18 months). In conclusion formation of vascular access by insertion of PTFE graft between profunda femoris artery and saphenous vein is an effective form of vascular access and merits consideration when primary forms have failed or not available.

Glomerulonephritis pattern in Sudan. Is it changing? *Suleiman S.M., Mukhtar B.I., Department of Medicine, U of K.* In this study, the renal biopsies done in between Jan. 1993—May 1997 were reviewed and classified according to age & histological pattern. Out of 350 biopsies results of 313 cases were found, 90 biopsies were from children below 16 yrs of age. It was found that the most common type is mesangiocapillary type in 81 biopsies, minimal change in 56, membranous and focal segmental in 40 each. These pattern is completely different from that seen in previous studies from the country.

Clinico pathological correlations in a group of African patients with membranous glomerulonephritis (MGN). *Youmbissi T.J., Mbakop A., Eloundou D., Departments of Nephrology and Pathology, Medical School, Yaounde, Cameroon.* Forty five Cameroonian patients in West Africa, diagnosed as having MGN were studied and followed up over a five year period. The mean age of these patients was 32.2 years with a female to male ratio of 5/4. Proteinuria (39%) and nephrotic syndrome (87%) were the two commonest clinical manifestations at the time of diagnosis. Seventeen cases (38%) were considered idiopathic while the rest was associated with known aetiological factors. Histologically the majority of the patients were either at stage I (35.5%) or stage II (37.8%) of the W.H.O. classification. Most patients in stage II and all the patient in stages III and IV showed associated important tubular, interstitial and vascular lesions. Immunofluorescence showed deposits to be mainly IgG (80%) and C3 (71%) while electron microscopy showed varied dense deposits in all cases. 17 patients with idiopathic MGN and 10 with Hepatitis B antigen associated MGN were followed up over 5 years. During this time 8 patients (30%) recovered completely, 11 patients (40%) improved their renal function, their degree of proteinuria and 8 others (30%) either were commenced on dialysis or died during the same period. All these 27 patients had undergone a 4 months period of oral steroid treatment.

Community based CAPD program at Soweto primary health care clinics. *Sefianou L., Milkov V., Butler O., Katz I.J., Baragwanath Hospital, Dept. of Nephrology.* Health care in our country is a scarcely achievable goal with the distribution of resources as they are. IPD has failed (no employment, no rehabilitation, high infection and mortality). However, CAPD is successful. CAPD is attractive as it is less centralised and allows greater flexibility. It is now the main focus of the Baragwanath renal programme and together with the help of primary health care clinics in the township we started a modified CAPD program in 1990 with 2 patients. CAPD trained nursing sisters from the 2 primary health care clinics serve to support this community based programme (CBP). Patients accepted for CAPD (by social worker, renal physician, renal nursing sister) have their Tenckoff catheter inserted at the hospital. On discharge they report to the primary health care clinic nearest to their homes for CAPD training. Dianeal solutions are delivered directly to the clinics and patients collect them. Some of the patients don't have running water or electricity therefore the technique was modified e.g. number and timing of exchanges, hand washing, treatment of peritonitis and follow up visits. The programme now comprises of 58 patients. Against the background of mass poverty, low socio-economic growth, massive unemployment. A CBP is a very attractive choice of dialysis for Africa. Future aims are to assess the

programme using peritonitis rate, quality of dialysis and success of transplants in these patients as markers.

The re-use of the dialyzers on chronic hemodialysis. *Hachim K., Zahiri K., Benghanem, Gharbi M., Fatihi E., Ramdani B., Zaïd D., Service de néphrologie, CHU Ibn Rochd, Casablanca, Morroco.* The re-use of the dialyzers after reprocessing is chosen recently by our unit. This process permits a control of the costs with great economic income, specially in our country. Our prospective study concerned the period from March to July 1996 about 24 hemodialysis patients (12 men and 12 women), their mean age is 47.62 years, 22 are jobless, let 91.67%, and all patients are under charge of donators. The dialyzers would be re-used 6.72 times on average after reprocessing on automatized apparatus using the sodium hypochlorite in 2.4% as sterilizing and desinfectant agent. The coagulation of the fibers has been the cause of rejection of the dialyzers in 92% of the cases. During 912 session of dialysis achieved during the study, we did not notice any febrile or naphylactoid reaction. The index KT/V for the re-used dialyzers represented 1.40 ± 0.21 without significant difference with this of the new dialyzers. The income is about 30 $US during the year, let 1279 $US for a patient during a year of treatment.

Hypertension in hemodialysis patients. *Hachim K., Benghanem Gharbi M., Fatihi E., Zahiri K., Ramdani B., Zaïd D., Service de néphrologie, CHU Ibn Rochd, Casablanca, Morroco.* The study turned about 36 hypertensive patients undergoing chronic hemodialysis. The hypertension is met with frequency of 46.8%, with an average of 40 years, and with male predominance. Most of these patients are base or average socio-economic level. The glomerulonephrotis have been the most frequent etiology of chronic renal failure (69.4%). The etiopathogenic of hypertension is multifactoriel: hemodynamic, hormonal and metabolic. . . . In the matter of treatment, we are recour to anti-hyeprtensor of 67% of the cases. The monotherapy was used in 44%, the bitherapy in 19% and the triple association in 3% of the cases. The conversing enzyme inhibitors were largely used in 33% of the cases: in monotherapy (16.7%), in bitherapy (13.9%) and in tritherapy (2.8%). The evolution was judged good with normalisation of the tensinal numbers in 80.6% of the cases. The complications detected are: left ventricular hypertrophy in 61.3% of the cases, coronariens insuffisancy in 22.2% of the cases.

Viral C hepatitis in hemodialysis patients. *Hachim K., Benghanem Gharbi M., Fatihi E., Zahiri K., Ramdani B., Zaïd D., Service de néphrologie, CHU Ibn Rochd, Casablanca, Morroco.* The objective of this study is to specify this affection according to the data of literature and after the study of 57 cases of viral hepatitis C among 160 patients who suffered fron chronic renal failure and periodically hemodialysed. The prevalence is 35.6%, let 57 patients: 27 men (45.64%) and 30 women (52.36%) based on search of the specific antibodies by Elisa's method. Among these 57 patients, 40 have been confirmed by Riba's method. The mean age of the patients is 45 years. The ancienty of the dialysis which the mean duration is 4.5 years and the number of transfusion which the mean number is 8.5 are the main risk factor of the disease. 56.41% of the patients developed a clinical symptomatology constitud, essentially, of asthenia, the asymptomatic form is noticed in 26.31% of the cases. Biologically, 47 of our patients had normal transaminases. The puncture biopsy of the liver achieved in 6 patients showed a chronical viral hepatitis in 2 patients. Even if our study concerns only one center, it permits to mind about this affection and must be completed in the future by a multicentric study.

Cytoskeletal proteins expression in experimental diabetic nephropathy (DN). *Chiwoneso Muchaneta-Kubara, Toru Sanai, Simon Oldroyd, Graham Thomas, Tarek Sobka, Meguid El Nahas, Sheffield Kidney Institute, Northern General Hospital Trust, Sheffield, UK.* The progression of diabetic nephropathy is associated with structural changes in the glomeruli, tubulo-interstitium and vessels. Cytoskeletal proteins such as α-smooth muscle actin (α-SMA), vimentin (V) and desmin (D) have been associated with mesangial proliferation, tubular injury as well as interstitial fibrosis. With that in mind, the changes in these proteins were investigated by immunohistochemistry, during the course of experimental DN (7, 15, 30, 60, 90, & 120 days) after the injection of streptozotocin (45 mg/kg) in groups of rats (n = 6). In normal rats, α-SMA and V were confined to vascular walls and glomeruli respectively with no positive stain for D. By contrast, in diabetic rats, α-SMA was detected in the glomeruli (3.2 ± 0.6%) and interstitium (2.1 ± 0.4) from day 7 onward and peaked on days 14 (11.4 ± 2.6%) $p < 0.001$ and 30 (3.2 ± 0.4%) NS, respectively. Vimentin also increased significantly by day 14 to 52.5 ± 11.9% compared with sham (23.3 ± 4.7%) $p < 0.05$, before returning to baseline on day 30. It also appeared in the peritubular capillaries, interstitial and tubular cells of diabetic rat kidneys from day 7 onward. Desmin appeared in the glomerular epithelial cells of diabetic rats from day 30 onward. Insulin treatment introduced on day 0 or 14 after DM, normalised blood sugar levels and reduced the expression of all the cytoskeletal proteins. Diabetic nephropathy leads to the neoexpression of cytoskeletal proteins in the renal cells.

Erythrocyte NA+ transports and endothelin-1 (ET) in chronic hemodialysis (CHD) patients. *Pedro L. Neves, M. Faisca, I. Bernardo, A.I. Anunciada, J.A. Ferreira, E. Viegas, H. Martins, A.M. Silva, Hospital Distrital de Faro. Centro de Hemodiálise de Faro—FMC. UCEH da Universidade do Algarve, Algarve, Portugal.* In the last years erythrocyte Na+ transport abnormalities have been described in uremia. It has also been reported that ET plasma levels are increased in uremic patients (pts.). In this study we analysed some erythrocyte Na+ transports: Vmax of Na-Li Countertransport (CT), Na-K-Cl Cotransport (CO) and Na-K Pump and the Na Leak-Kpna (Na+ concentrations evaluated by atomic emission spectrophotometry), in two groups of subjects: I—Control (n = 18) and II—CHD pts (n = 40). Several biological and laboratorial parameters were analysed, including the plasma ET levels (RIA—Phoenix Pharmaceuticals). Concerning the erythrocyte Na+ transports G-II showed only a decreased Vmax of the Na-K-Cl CO (269 ± 17 vs 342 ± 31 μmol/L cel h-1, $p = 0.029$), reflecting probably the presence of bumetanide-like factors. The ET plasma levels were significantly higher of CHD pts (n = 24) than in control subjects (n = 9) (9.25 ± 1.0 vs 1.89 ± 0.2 pg/100 ml, $p < 0.001$), and we found a positive correlation in these pts, between ET plasma levels and the Vmax of the Na-Li CT ($r = 0.514$, $p = 0.010$). It has been found that the Na-H exchanger is stimulated by ET. Our results show that in CHD pts, the Vmax of Na-Li CT (an operating mode of the Na-H exchanger), can be related to ET plasma levels.